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EPONYMS.

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XXII. COCK'S OPERATION.

THE description of Cock's operation is to be found in the twelfth volume of the third series of the *Guy's Hospital Reports* for the year 1866. It is a short paper entitled "A few words on the means to be adopted for establishing a communication between the Bladder and the exterior of the Body when the urethra has become impermeable. The last resource available in certain cases". Mr. Cock says:—

"In this short communication I have no intention of alluding to the treatment of ordinary stricture; nor shall I yield to the temptation of criticising the various methods and appliances which, within the last few years, have been either invented or resuscitated for the purpose of restoring a narrowed urethra to its normal condition. Some ingenious mechanical contrivances cut, some tear, some burst, some are supposed to dilate with marvellous rapidity. My objection to their use is founded on a forty years' experience, which has taught me that such cutting, tearing, bursting, or rapid dilatation is often exceedingly mischievous in its effects, and fails in establishing a permanent cure, and that the object in view may be accomplished by much milder, surer and safer means.

"The purport of this paper is not to show how a strictured urethra may be treated, but how the bladder may be got at when the canal has become positively and absolutely impermeable to instruments of every description.

"A class of cases not unfrequently comes under our observation, where the bladder must be got at somehow to save the life of the patient and where it cannot be reached, either by ordinary catheterism through the urethra, or by tapping above the pubes or through the rectum. To remedy this complication of evils—viz., an impermeable urethra; a disorganised perineum, of which all, or nearly all the anatomical features have disappeared; fistulous sinuses communicating with the urethra, and distilling urine; recurrent abcesses from the irritation of the extravasated fluid; broken down health from diseased bladder, the forerunner of renal disorganisation; I say, to give

the patient a chance of life or restoration, it is necessary to establish a free communication between the bladder and the exterior.

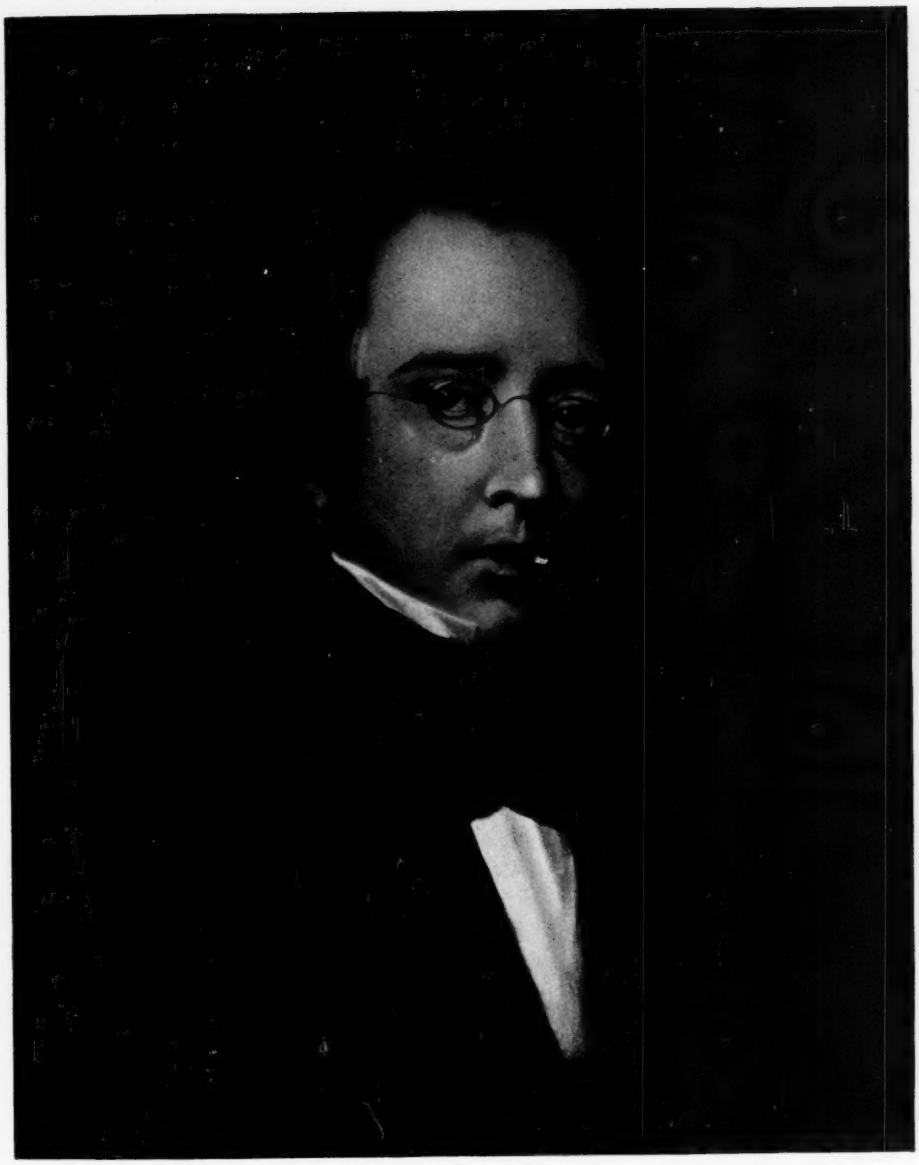
"The natural passage through the urethra is not available, as the channel has long been either rendered impermeable or altogether destroyed. The sound and catheter are therefore useless; while the contracted condition of the bladder forbids the operation of puncture behind the prostate.

"The surgical means adopted in these miserable cases should have a twofold object in view—an immediate and a more distant result. The one object is the prompt establishment of a free communication with the bladder behind the obstruction. The second and more remote is the restoration of the urethra to its normal condition, so far as this may be possible. For many years I have adopted an operation which is at once more easy, simple and successful than those which are in general use at the present time. However complicated may be the derangement of the perineum, and however extensive the obstruction of the urethra, one portion of the canal behind the stricture is always healthy, often dilated, and is accessible to the knife of the surgeon. I mean that portion of the urethra which emerges from the apex of the prostate—a part which is never the subject of stricture, and whose exact anatomical position may be brought under the recognition of the finger of the operator.

"Thus, when we cannot introduce a catheter by the ordinary method, and even when we cannot tap the bladder through the rectum, it still remains to us to tap the urethra as it emerges from the prostate, and thus to effect the desired communication. The only instruments required are a *broad* double-edged knife, with a very sharp point; a large silver probe-pointed director with a handle; and a canula, or a female catheter modified so that it can be retained in the bladder.

"The patient is to be placed in the usual position for lithotomy; and it is of the utmost importance that the body and pelvis should be straight, so that the median line may be accurately preserved. The left forefinger of the operator is then introduced into the rectum, the bearings of the prostate are carefully examined and ascertained and the tip of the forefinger is lodged at the apex of the gland. The knife is then plunged steadily but boldly into the median line of the perineum, and carried on in a direction towards the tip of the left forefinger, which lies in the rectum. At the same time by an upward and downward movement, the vertical incision may be carried in the median line to any extent that is considered desirable. The lower extremity of the wound should come to within about half an inch of the anus. When the operator has fully assured himself as to the relative positions of his finger, the apex of the prostate, and the point of his knife, the latter is to be advanced with a motion somewhat obliquely either to the right or the left and it can hardly fail to pierce the urethra. If, in this step of the operation, the anterior extremity of the prostate should be somewhat incised, it is a matter of no consequence.

"The knife is now withdrawn and the left forefinger is still retained in the rectum. The probe-pointed director is carried through the wound, and, guided by the left forefinger, enters the urethra and is passed into the bladder. The finger is now withdrawn from the rectum; the left hand grasps the



EDWARD COCK

1805 - 1892

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director, and along the groove of this instrument the canula is slid until it enters the bladder.

"The operation is now complete, and it only remains to secure the canula in its place with four pieces of tape, which are fastened to a girth round the loins. There will probably be no escape of urine until the stilette is removed from the catheter."

The operation used to be performed with great success by Mr. Cock, who was a consummate anatomist with many years training as a teacher in the dissecting room at Guy's Hospital. It proved less satisfactory when it was undertaken by other surgeons. It gradually fell into complete disuse, being replaced by less dangerous methods. It served its purpose, however, for it displaced the practice of tunnelling the prostate—a terrible operation undertaken in cold blood and without anæsthesia. The surgeon took "a small but strong catheter, one that will not easily bend; and firm of purpose with unflinching hand, and fearless—I had almost said remorseless—heart, he carried it through all intervening tissues and obstacles into the bladder. It is as it were puncturing the bladder with a blunt instrument—tunnelling through the perineum under cover of the urethra. It is neither scientific in its conception, surgical in its performance nor anatomical in its details." Yet it lingered on, certainly until after the year 1880, for as a dresser it was my duty to support the unhappy patient as he sat shivering at the edge of a bed whilst the surgeon 'cured' the stricture. It is no wonder therefore that patients did not apply for relief until the results of the stricture had become unbearable and the perineum a mass of scar tissue or riddled with sinuses. It is largely due to the Guy's teaching that milder methods came into use by gradual dilatation and the employment of less rigid catheters.

'Old Cock' or 'Teddy Cock', as he was called affectionately by many generations of Guy's students, was one of the best beloved of their surgeons. In appearance he was a man rather below the average height, but always with a slight stoop which made him seem prematurely old. His grand head and face and pleasant expression, however, atoned at once for a somewhat diminutive frame. His delicate pink complexion; his blue eyes protected by gold-rimmed spectacles; his firm, well-chiselled, aquiline nose; and a most attractive smile, created an impression not easily forgotten. His white hair remained thick to extreme old age, and curling up at the end gave him somewhat of a gallant air, whilst his short beard being confined to lips and chin, left the dimples of his rounded cheeks exposed in laughter. A few words with him and you found that you were in the presence of a man of high culture possessed of an infinite sense of humour.

In dress he was peculiar and quite indifferent to all conventional ideas of professional attire. In summer he usually wore a large shepherd's plaid for trousers, a buff waistcoat with onyx buttons, and a silk-lined jacket, with a blue-spot necktie and low-crowned soft wide-awake hat. In winter the white hat was changed for a black one of the same shape and the silk fly-jacket was replaced by a pilot coat. A massive gold chain, a silver snuffbox, and an ivory-handled cane completed the picture. His peculiar attire was probably that which experience had taught him was most comfortable to

wear when on horseback, for Cock began to practise when consultants as well as general practitioners rode to see their patients.

Born in 1805, he died in 1892. He was a nephew, by the marriage of his aunt, to Sir Astley Cooper, and was apprenticed to his uncle at the age of 16. Uncle and nephew were akin in their love of fun and practical joking, so that the master and the apprentice were well-assorted. The master loved Cock and the apprentice became a hero-worshipper. In 1825, whilst Cock was still bound by his articles, Sir Astley Cooper resigned his lectureship on surgery at the United Borough Hospitals of St. Thomas's and Guy's, in favour of his nephew Bransby Cooper. The nomination was not accepted. Mr. South was appointed, and feelings ran so high that Guy's Hospital decided to establish a school separate from that of St. Thomas's. Bransby Cooper was appointed Lecturer on Anatomy and Mr. Cock was chosen Demonstrator of Anatomy, although he was not yet qualified and was only twenty years of age. He was admitted a Member of the Royal College of Surgeons in 1828 and was selected as one of the first twenty-seven Fellows of the College when that order was established in 1843. He worked assiduously in the dissecting rooms as Demonstrator until 1838, when he was elected Assistant Surgeon and in 1849 full Surgeon to the hospital. He remained a bachelor until 1870, when he married Miss Nunn, with whom he lived most happily until her death in 1886.

The greater part of his active life was spent in St. Thomas's Street within a few yards of Guy's Hospital, and of his doings here innumerable stories were told, some true, some *ben trovato*, but all indicative of the sociability and genuine benevolence which made him beloved alike by students and patients. At the back of his house was a 'garden' of about twenty feet square. It had a few inches of shabby grass with a fountain in the middle, the home of one eel; a fig tree, a shrub which in allusion to the speciality practised by Mr. Cock the students called 'the copaiba'; and a garden seat. An old gardener came as an out-patient to the hospital whose face was so destroyed by a rodent ulcer that no one would employ him. Mr. Cock provided him with a new nose kept in place by a pair of spectacles, and engaged him to look after his 'garden'. The man entered into the spirit of the thing, whetted his scythe, mowed the grass, made paths and borders on a microscopic scale, planted flowers in full blossom, and received his wages in due season. Master and man were alike happy so long as the illusion lasted.

Mr. Cock, like Charles Lamb, had a most engaging stutter which lent piquancy to the stories told by him or invented of him, just as Spoonerisms have been invented in these later years. It is said, probably without a shadow of truth, that the constable on night duty once found him sitting with his back to the wall facing his own house. The constable, who like all members of the force stationed near the hospital was on friendly terms with him, said, "What are you doing there, Sir"? To which Mr. Cock replied, "Waiting for Mr. C-C-C-Cock's house to come round."

Once when he was engaged at out-patients he told his class that only stupid and careless people had their pockets picked. When he had seen the last out-patient he felt for his snuffbox and found that it had vanished. He

went away saying what he thought in the highly-spiced vernacular of the period, and found a patient waiting to see him when he got home. Cock examined him, prescribed, and instead of a fee the man presented him with the missing snuffbox, saying that he had called to return it. " But, how on earth did you do it ? " said Cock. " Oh, it's very easy when you know 'ow " replied the man, who then proceeded to admire the pictures on the wall, the beauties of which were explained to him. He then said 'Good-bye', and as he was being shown out he handed back the snuffbox which he had taken for the second time.

There are two portraits of Mr. Cock. The one here reproduced, to which my attention was called by Mr. John Everidge, O.B.E., F.R.C.S. Eng., shows him as a young man. The other, painted much later in life, hangs at the top of the Grand Staircase at Guy's.

ATROPHIC DISEASE OF THE SHOULDER-JOINT.

By J. P. ROSS, London.

INTRODUCTION.

Atrophy of the humerus progressing to complete disappearance of the upper end of the bone is of sufficient rarity and interest to warrant thorough investigation. The Museum of the Royal College of Surgeons contains a good specimen of atrophy of the head of the humerus, but the shaft appears unaffected (*Fig.* 120). This specimen was presented by M. Charcot, who described it in his *Leçons sur les Maladies du Système nerveux.* He used it to illustrate the rapidity with which bone destruction may take place in locomotor ataxia; for he had been able to note the time of onset of the arthropathy, and at the end of three months the head had been destroyed.

Fig. 120.—Upper end of humerus presented by Charcot. (*No.* 1022.1, *R.C.S. Museum.*)

Charcot in his original description[1] of the peculiar arthropathy which occurred only in association with disease of the nervous system dealt merely with the clinical aspects. He gave a vivid account of several cases and showed that, in his experience, the joint lesions of tabes dorsalis first appeared at a definite period in the history of the disease, when motor incoordination was added to lightning pains. Thus, although they may be an early manifestation of tabes, they are never the first evidence of involvement of the spinal cord. He made no attempt at this time to evolve theories with regard to the nerve cells or fibres involved.

In a later publication[2] he gave further descriptions of these joint lesions, dividing them up for purposes of classification into those with painful effusion occurring in Pott's disease, injury to the spinal cord, myelitis, and hemiplegia due to cerebral softening; and tabetic arthropathy in which at a definite period in the course of tabes sudden swelling of a whole limb appeared, which was later confined to one of its joints, the whole process being painless and afebrile. He added an account of the morbid anatomy of the first type, emphasizing the hyperæmia and thickening of the synovial membrane of the joint and neighbouring tendon sheaths, and stating that arthritis of neurotrophic origin might be distinguished from rheumatoid arthritis by limitation

of the lesions to joints of paralysed limbs, the sudden onset at a definite period of the disease, and the co-existence of other lesions of a like nature, such as trophic sores, muscle atrophy, and cystitis.

The arguments brought forward in favour of the trophic origin of the joint lesions of tabes dorsalis were: (1) That there is no evidence of a traumatic or rheumatic factor; (2) That the arthropathy appears at a constant and early stage of the disease of the nervous system; (3) That the morbid anatomy and course of the disease are quite unlike those of any joint affection of an inflammatory nature; and (4) That atrophy of the anterior horn cells is to be found in cases showing arthropathy, this atrophy not being attributable to disuse of the muscles, since it differs from the changes in the anterior horns corresponding to an amputated limb.

The grey matter of the spinal cord is frequently affected in tabes, usually the grey matter of the posterior horns. But if the anterior horns are involved, trophic disturbances in the limbs result. Another piece of corroborative evidence is that arthropathy has been noted in chronic anterior poliomyelitis.

In the same lectures Charcot gave some consideration to the theories brought forward to explain neuropathic arthritis. His teaching may be summarized by saying that he considered the vasomotor theory insufficient to account for the lesions, his conclusion being that vasomotor palsy or spasm alone was not capable of producing trophic disturbances. The alternative theory, which postulated the existence of trophic nerves, he believed to be more acceptable, in spite of the lack of anatomical confirmation.

The early incidence of trophic affection of joints in certain lesions of the central nervous system is now well recognized, and it is taught that cases of tabes may be met with in which joint disease is present before any other sign of nerve disorder can be detected.[3,4] It is unusual, however, for the joint disease to reach an advanced stage of development before obvious signs of nervous disease may be found on clinical examination; and the present case is being placed on record because the joint under consideration had been abnormal for ten years and grossly deformed for three years before signs of disease of the nervous system were discovered.

DESCRIPTION OF CASE.

E. H., a married woman, age 45, was admitted to St. Bartholomew's Hospital under the care of the Surgical Professorial Unit in January, 1925, with the following history.

Early in 1914 she fell downstairs and injured her right shoulder. She consulted a medical man, but the condition did not demand hospital treatment or even the wearing of a support. In 1917 she noticed swelling of the right upper arm and some loss of power, which recovered under electrical treatment in three weeks. Again, in 1921, she had to give up her work—housework and washing—on account of a return of pain, swelling, and loss of power in the right arm. At that time she attended the hospital as an out-patient; her shoulder was X-rayed (*Fig.* 121), and a diagnosis of Charcot's disease of the shoulder-joint was made. Signs of disease of the nervous system were not detected, and after a short period of palliative treatment she

was able to resume her work and did not re-appear at the hospital until the time of the present admission.

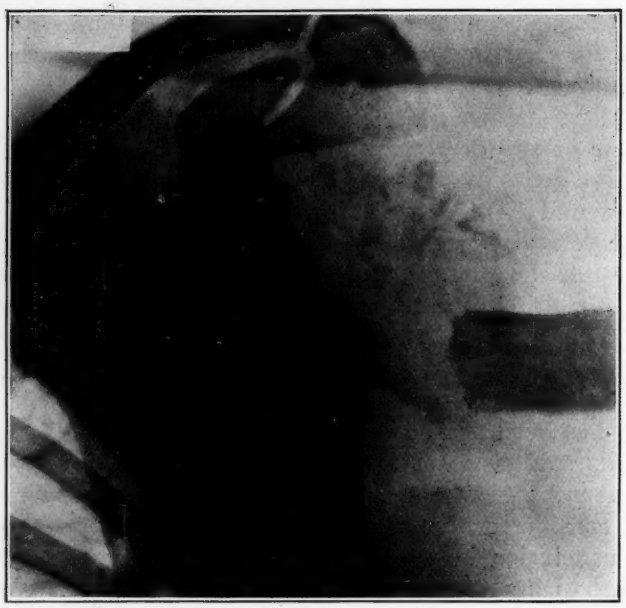
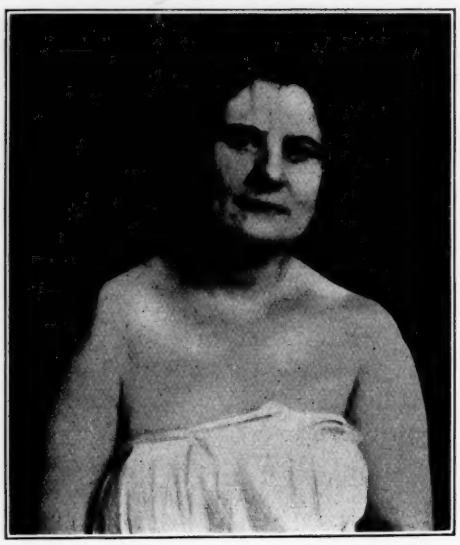


FIG. 121.—Skiagram of right shoulder-joint. April, 1921.

In the interval between 1921 and the end of 1924 the condition of the arm did not worry her, her grip improved, and she was able to wash and wring clothes. Towards the end of 1924, however, she was worried by weakness of the right arm, pain in the palm of the right hand, numbness of the right shoulder and upper arm, and slight dragging of the left leg. Apart from the disability in the right arm she had not suffered from any illness in the past. She had seven healthy children, and one miscarriage between the first two children. She had never suffered from any form of joint disease.



FIGS. 122, 123.—Showing the deformity of the right shoulder and the narrowing of the palpebral fissure on the right side.

Examination of the patient revealed great deformity of the right upper arm. There was a marked hollow below the acromion, and the head of

the humerus could not be palpated (*Figs.* 122, 123). Active movement of the shoulder was practically absent, the patient being able to get about 30° of abduction and 15° of anteroposterior movement; the joint could be moved passively very freely in all directions, soft crepitus being detected. There was only half an inch of wasting of the arm. The length of the arm varied within a range of nearly three inches according to the state of contraction of the deltoid muscle. The right hand was slightly 'clawed' and the interossei were wasted; the skin was noticed to be coarse, but no trophic lesions were found. Movements of the right elbow and wrist were full and strong, and signs of disease could not be detected in any other joint. A considerable degree of scoliosis was present.

There were definite signs of involvement of the spinal cord in the cervical region. They were paresis of the right cervical sympathetic, definite hypalgesia of the right side of the face, neck, and arm, and impairment of thermal

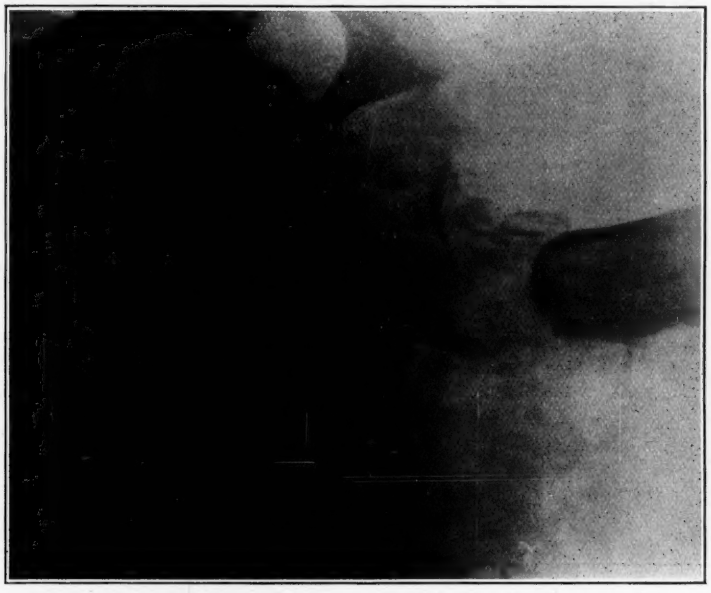


FIG. 124.—Skiagram of right shoulder-joint. October, 1924.

sense over the right shoulder and arm. Vibration sense was lost in the right arm. There was slight hypæsthesia of the right face and right arm, but this was not nearly as marked as the loss of pain and temperature sense. The left leg showed muscular weakness, there was exaggeration of the left knee- and ankle-jerks, and the left plantar response was extensor. The patient showed no signs of syphilis, and the Wassermann reaction in the blood was negative.

Skiagrams of the joints showed that the head and neck and upper part of the shaft of the humerus had disappeared almost completely. The first skiagram (*Fig.* 121) was taken in 1921, and it is interesting to compare it with the skiagrams (*Figs.* 124, 125) taken in 1924. Apart from the fact that the upper end of the shaft of the humerus had become smoother and more rounded

off, there was very little X-ray evidence that the lesion in the shoulder-joint was progressing, the other bones and bony abnormalities being almost unchanged in the interval. Besides the absorption of the head and part of the shaft of the humerus the changes in the scapula must also be carefully studied. Attention is drawn to the enlargement of the glenoid, which appears to be even shallower than normal, and to the new bone laid down at and below its inferior border, to which reference will be made later.

COMMENTARY.

In attempting to arrive at a diagnosis of the condition the possibilities which had to be considered were :—

1. That the humerus had been fractured at the time of the original accident, and that the subsequent atrophy of the head and upper part of the shaft was due to interference with nutrition brought about by the fracture.

2. That the condition was a manifestation of the atrophic type of arthritis deformans.

3. That the condition was a neuropathic arthropathy.

FIG. 125.—Skiagram of right shoulder-joint. October, 1924.

If the first or second possibility was to be accepted as the correct solution of the problem, then the signs of nervous disease which were discovered at the last examination would have to be taken to indicate an early stage of some intercurrent nervous disease having no relationship to the joint affection.

1. **Trauma.**—As far as the possibility of a fracture having been the original cause is concerned, it would appear that the patient's own history of the injury is the best argument against its being the correct one. She stated that, although she fell heavily and bruised her shoulder severely, yet she was not disabled for any appreciable length of time, being able to carry out all her work without pain a few days later; and she never had to wear a sling or any form of splint after the accident.

It is interesting to note, however, that a history of an accident too severe to be entirely disregarded as a causal factor is sometimes obtained in cases of undoubted neuropathic arthropathy. As an example of this one may cite the case, recorded in Marsh and Gordon-Watson's *Diseases of the Joints and Spine*, of "a hawker who sustained an injury (probably rupture of some

muscular fibres) to his shoulder one year previously, followed by a swelling which gradually subsided. Four months later a sudden painless effusion occurred in the same joint, and two months afterwards well-marked grating was observed". This patient showed signs of syringomyelia. It may be that the injury determines the particular manifestation of the disease of the nervous system, and this supposition receives support from animal experiments carried out by Eloesser,[5] who was able to show that typical neuropathic arthropathy could be produced by inflicting injury on a joint in a limb which had been deprived of its sensory nerve supply by previous division of the corresponding posterior roots.

It was suggested that a fracture near the head of the humerus might lead to absorption of the upper end of the bone in the same way that absorption of the head of the femur may follow fracture of its neck. A good example of this condition is afforded by another case recently under the care of the Surgical Unit at St. Bartholomew's Hospital.

The patient was 5 years of age, and it was stated that when 6 months old she fell out of her perambulator and injured the left hip. Two weeks later a skiagram showed a fracture through the neck of the femur, and the limb was put up in plaster. The plaster was changed every three months, and in the course of four and a half years her lesion came to be regarded as tuberculous arthritis of the hip-joint.

On examination, flexion and extension were found to be free at the left hip, but abduction was limited. The head of the femur could not be palpated. The left leg was considerably wasted and shortened. There was no evidence of old inflammatory mischief in the joint. Skiagrams showed absence of the head and neck of the femur, the top of the shaft articulating with a flattened false acetabulum (*Fig.* 126). Within a few days she learnt to walk without pain, and was sent home wearing a caliper splint.

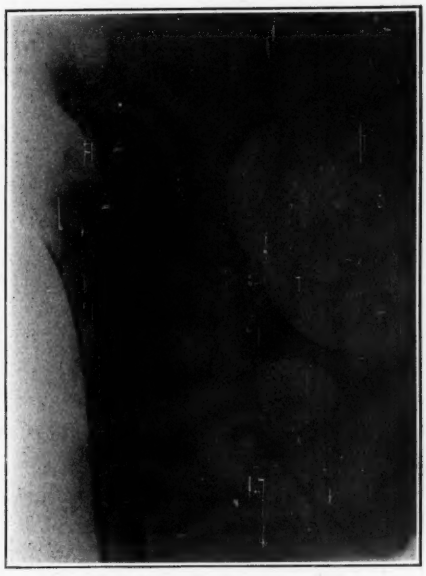


FIG. 126.—Skiagram of left hip-joint (posterior view), showing absorption of the head and neck of the femur.

The collection of Nubian bones recently added to the Museum of the Royal College of Surgeons contains several specimens of femora from which the head and neck have been worn away, and it is believed that in these also the condition can be explained by fracture of the neck of the bone with subsequent atrophy and absorption of the proximal fragment.

There is no reason to suppose that the head of the humerus is comparable to the head of the femur in this respect. It is possible for small fragments of bone torn off the upper end of the humerus to become absorbed and disappear, but I can find no reference to any case of absorption of the whole head of the humerus after fracture through the neck.

2. **Arthritis.**—In considering the second possibility, that the joint lesion might be a manifestation of arthritis deformans, there are several points

which can be made in its favour. Injury is frequently associated with the onset of this disease. Also, in arthritis deformans of the shoulder-joint the deformities of the glenoid may be very similar to those seen in this case—enlargement of the fossa, which becomes shallower than normal, with ossification extending downwards into the long head of the triceps muscle. It is well known that rarefaction of the cancellous tissue at the end of a long bone may take place to such an extent that bending of the bone results; but cases of actual loss of considerable parts of the bone are rare, though dislocation due to bone loss, and not resulting from stretching of peri-articular structures, has been recorded.

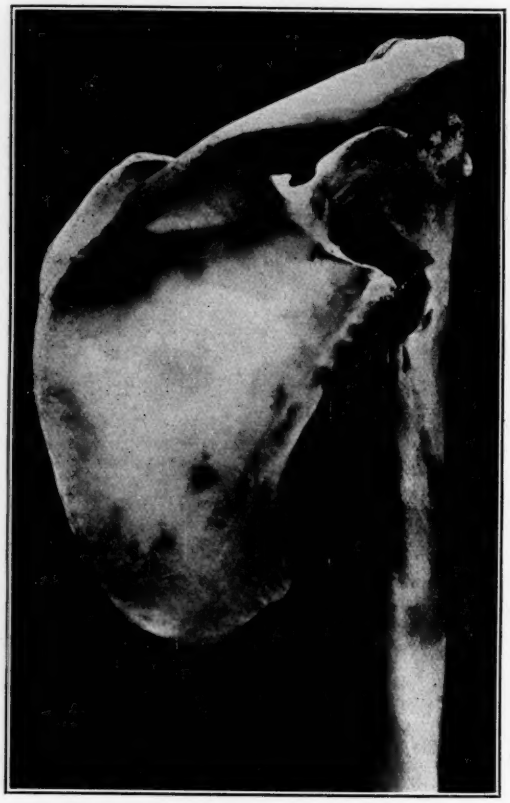


FIG. 127.—Shoulder-joint. (*No.* 1922, *R.C.S. Museum.*)

In this connection reference may be made to a specimen in the Museum of the Royal College of Surgeons (No. 1922), presented by Mr. Jonathan Hutchinson, and described in the catalogue as follows:—

"The bones of a right upper extremity. All the bones appear lighter than normal, the scapula especially being unusually thin and transparent. The glenoid cavity and the head of the humerus have been completely absorbed. The tuberosities alone remain. Upon the inner surface of the shaft of the humerus, just below the tuberosities, is a concavity, directed from above downwards, and which has extended half through the bone so that the medullary canal is widely exposed. The concavity has apparently been produced by the friction of the displaced humerus against the semilunar concavity of the scapula upon which it moved" (*Fig.* 127).

An abstract of the clinical history is appended, from which it appears that the patient was aged upwards of 60, emaciated, and bedridden. The shoulders, elbows, wrists, knees, and interphalangeal joints were affected symmetrically. There was a deep hollow under each acromion, and the head of the humerus had evidently been absorbed to a great extent and displaced towards the axilla. A description of the other joints is given, and then it is stated that there was not the slightest swelling of the parts adjacent to the joints, that there was great wasting of the limbs, but the fingers looked quite natural. The malady began in the patient's shoulders and gradually progressed. She suffered great pain in the joints, though movement gave no pain. Her pupils reacted to light, and she had no bladder symptoms. The malady

was believed to be "chiefly one of general rheumatism", and it seems possible that it may have been one of the rare instances of arthritis deformans associated with marked bone destruction.

In the case at present under consideration there are several points against the diagnosis of arthritis deformans. Pain and limitation of movement are the rule in arthritis deformans, whereas the outstanding features in the present case are weakness and abnormally free mobility. Also, if those rare cases referred to above be left out of consideration, the common change in the humeral head in arthritis deformans is enlargement, lipping, and flattening. In short, abnormal mobility and actual loss of bone, both of which are present in this case, are two of the important features differentiating a neuropathic joint affection from arthritis deformans.

3. **Neuropathic Arthropathy.**—By a process of exclusion, therefore, as well as by clinical examination of this case, one is forced to the conclusion that the condition of the shoulder-joint is neuropathic in nature. This is confirmed by descriptions given of specimens and of other cases in which disease of the joint was proved to be a manifestation of a lesion of the nervous system.

With regard to the specimen presented to the Royal College of Surgeons by Charcot (*Fig.* 120), the Museum catalogue states :—

"The head of the bone has been completely destroyed, the articulating area formed by the upper end of the shaft being slightly convex, and for the most part smooth and eburnated. Patches of new bone have been produced on the external aspect of the shaft and elsewhere in the neighbourhood of the new articular surface. The compact wall of the shaft is thinned by atrophy."

In the above description no mention is made of the state of the glenoid, but it is stated elsewhere that in Charcot's disease "the glenoid cavity is replaced by a large articular hollow bounded above and in front by the acromion and the coracoid process and below by a mass of new bone springing from the axillary border of the scapula and produced apparently by ossification of the long head of the triceps" (Marsh and Gordon-Watson). This mass of new bone can be seen well in the skiagrams of the present case (*Figs.* 121, 124).

Charcot's specimen described above was obtained from a shoulder in which the disease was only moderately severe. It is usual for these lesions of the shoulder-joint to give the appearances characteristic of the atrophic type of Charcot's disease, with much effusion in the early stages, and extensive bone destruction later. Dujarier[6] summarizes his experience by saying that, besides the head of the bone, frequently several centimetres of the diaphysis are absorbed, and it is possible to introduce several fingers between the prominence of the acromion and what remains of the upper end of the shaft of the humerus. He illustrates his article in Le Dentu and Delbet's *Traité de Chirurgie* with a photograph showing advanced bilateral tabetic arthropathy of the shoulder-joints, each joint appearing in the picture to be as extensively diseased as the shoulder now being described.

It is possible for various lesions of the central nervous system to produce such trophic changes in the shoulder-joint; and in this case the clinical signs, slight though they are, suggest syringomyelia in the cervical region of the

spinal cord as the most likely cause. The shoulder is the joint most commonly affected in syringomyelia, and the sensory impairment, the wasting of the muscles of the hand, the cervical sympathetic paresis, the pyramidal tract involvement, and scoliosis may all be taken to be manifestations of this disease. It is stated that syringomyelic joint affections are more common in males because men are more exposed to injury than women. In this case, however, there is the history of trauma severe enough to justify the belief that it is an exception to the general rule. The loss of a considerable length of humerus and the tendency to ossification in the triceps muscle are recognized as characteristic appearances in syringomyelia.

The time of onset of arthropathies in the course of syringomyelia varies considerably in different cases, and it is stated by Dujarier that, although as a rule well-marked signs of nervous disease precede the joint manifestations, yet the latter can be the first sign of disease, as in the case under discussion. The same author also quotes examples showing that the arthropathy of syringomyelia may progress for twenty, thirty, or even thirty-five years.

TREATMENT.

The only treatment worthy of consideration would be an operation planned to give greater stability to the shoulder by fixation of the joint. Consideration had to be given to the fact that the woman had fairly good function in the right arm and was able to do her housework, and even to wash clothes in spite of the undue mobility and weakness of the shoulder. The increased risk of suppuration and of delayed and imperfect healing owing to the trophic affection of the tissues was taken to contra-indicate operative treatment. Also it had to be borne in mind that weakness of the left leg was becoming a more troublesome feature. This evidence of spreading involvement of the cord was a further argument against treatment of the shoulder itself.

The patient has been examined from time to time since the beginning of 1925, and the decision not to operate on the joint has been justified by the subsequent course of the case. The shoulder remains practically unaltered, but the legs are becoming progressively weaker.

I wish to acknowledge my indebtedness to Professor Gask for permission to publish these cases; to Sir Arthur Keith for allowing me to make full use of the specimens in the Royal College of Surgeons Museum; and to Miss Vaughan, of the Dunn Laboratories, for her help with the illustrations.

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INTRACEREBRAL CALCIFICATION.

By JOSEPH J. LEVIN, Johannesburg.

Intracerebral calcification is a relatively rare condition—so rare, in fact, that when Heuer and Dandy made an exhaustive study of the subject in 1916 there had been only seven cases previously reported. I can find no mention of such a case in any of the common text-books of surgery. Since Heuer and Dandy's publication in 1916 a few more cases have been published by different authors, but I think I am right in saying that not more than twenty or thirty cases of intracerebral calcification, definitely proved either by operation or autopsy, have been reported. As far as I can ascertain from the journals which are at my disposal, and from correspondence with Mr. D. M. Greig, Conservator of the College of Surgeons Museum, Edinburgh, not one of these cases is an exact counterpart of my first case, and of what I believe my second to be.

E. J. Heuer and W. E. Dandy[1] say they have found only twenty recorded cases of calcification of tumours. A tumour recognized by X rays, described by A. Church[2] in a boy of 15, was found *not* calcified. Calcification of an aneurysm is recorded by Heuer and Dandy.[3] J. W. Ogle[4] records calcareous and bone formation, but no case of a hæmatoma apparently. Probably most of his 14 cases were of healed tubercle; at any rate there was pulmonary tubercle in 6. Only one case had recurring epilepsy. The same author[5] records the case of a male, age 35, who had had pain in his head for two years after having had his arm broken in a fall. Later, he frequently vomited, became very tremulous, and then had dysphagia for a week, and died with stertorous breathing soon after admission to hospital. The post-mortem examination showed the dura in the middle fossa puckered and thickened, brick-red in colour, and adherent to the brain. Underlying this on each side the brain was a small cavity containing hæmatin crystals. The case is not too fully described, but was evidently a hæmatoma following injury. There was no note of the cysts having calcified walls.

L. Rokitansky[6] writes of the formation of blood cysts following injury. "The walls of the sac, especially that one which adheres to the dura mater, sometimes become the seat of ossification, that is, of bony concretions in the form of plates." The word 'ossification' in those days was often used instead of 'calcification'.

J. T. Murphy publishes the case of a primipara, age 47, who had convulsions affecting only the right side two to four times yearly during seven years. A radiogram showed a barrel-shaped mass which he considered a calcified cyst. He gives a few references,[7] but none of a calcified hæmatoma.

E. A. Miller[8] records the case of a man, age 60, who had recurring epilepsy during twenty-six years, with pain in the left parietal region. The twitching began in the right hand and affected the right side of the body.

The calculus was removed successfully by operation, and the organic basis was 'decomposed' blood. There was no history of injury, but that seems to me to be the most likely source of such an affection.

John O'Sullivan,[9] in an article entitled "Some Rarer Intracranial Calcifications and Ossifications," classifies calcifications into four groups, namely: (1) Symmetrical retro-bregmatic ossifications of the dura; (2) Calcified cysticercus; (3) Calculi in the brain; (4) Calcified tumour.

None of his cases or groups corresponds exactly to my cases. In some there is a history of injury. A skiagram of the case in the fourth group, figure 15, is somewhat similar to that of my first case, but the pathological report of his specimen is entirely different. He states that the clinica history and findings, together with the X-ray examination, lead to the conclusion that the war injury was *not* responsible for the patient's condition. This case, he says, was probably a psammoma.

Of my own two cases, one was referred to me by Dr. Harte, and the other by Drs. Heimann and Campbell. The diagnosis of the first case was proved conclusively by successful operation. In the second case, as the symptoms were by no means inconveniencing the patient, I did not feel justified in operating on her, and therefore have not so far proved the diagnosis conclusively. The history, however, is so exactly like that of the first case, and the skiagram so suggestive, that one has come to the conclusion that the nature of the mass is the same.

REPORT OF AUTHOR'S TWO CASES.

Case 1.—This case was shown at the monthly meeting of the Witwatersrand Branch of the British Medical Association in October, 1925. The history is as follows:—

The patient, an Indian, age 21, who resides in Standerton, consulted Dr. Harte on July 20, 1925, complaining of headache and attacks of 'shivering' of the right arm and leg.

He is a tailor by trade, is married, and has two healthy children, age 2 years and 2 months respectively.

Personal History.—His father states that he came to South Africa between the ages of seven and eight, that he went to school but was not very clever there. He learned to read and write English, but has forgotten that art; he can, however, read and write his own language. He is clever as far as money matters are concerned! In 1910, when he was 6 years old, he had a fall and sustained a fracture of the right forearm and also an injury to the *right side of his head.* About ten days after the accident he felt faint and vomited. He had a similar attack twelve months later, and these attacks became more frequent as time went on, recurring every six months and lasting only a few minutes. Before each attack he complained of seeing 'double'. About three years ago, in 1922, during one of these attacks his *right* hand began to 'shiver', and his fingers opened and closed involuntarily. The 'shivering' spread up his arm, then down the *right* side of his body to the thigh, leg, and toes. Both the upper and the lower limb performed constant movements during the attack. The attack lasted seven or

eight days; he slept very little during the period, but when he did sleep the movements of the limbs stopped. Three months later he experienced a similar attack, and noticed that a few days beforehand he had pain in both ears and was slightly deaf, and that a tingling in his right fingers preceded the attack. Since then he has had similar attacks about every three months, lasting from three to eight days.

About two years ago, whether during one of these attacks or not we cannot ascertain, he has the history of a severe headache accompanied by blurred vision and aphasia (he understood what was spoken to him and he could speak, but his friends did not know what he was talking about—jargonaphasia). This lasted one week, and he has had similar attacks since.

FIG. 128.—*Case* 1. Skiagram showing the calcified patch in the left temporoparietal region.

During all these attacks the 'shivering' was confined to the *right side of the body*; he was never completely unconscious. There is also a history of three bouts of vomiting during June, the month before coming under Dr. Harte's observation. His father states that he had some treatment for this condition before 1922. Since 1922 he has been constantly under treatment by three or four doctors.

FAMILY HISTORY.—Nothing to note. His father and mother are alive and apparently healthy.

OPHTHALMOLOGIST'S REPORT.—"Vision, tension, fundi, and extra-ocular muscles are normal. Pupils slightly dilated, but react normally. Wernicke's hemiopic pupillary reaction is negative. The field of vision shows a right homonymous hemianopsia.

"His cerebration is slow, and his general intellectual capacity is diminished. His perception of superficial and deep-seated sensibility is normal. His stereognostic sense is normal. He has no apraxia, either ideologic or ideomotor. He has no aphasic phenomena of any form."

RADIOLOGISTS' REPORT.—An X-ray photograph (*Fig.* 128) was taken by Drs. Steuart and Olivier, who reported as follows :—

"There is an irregular calcified patch in the left temporoparietal region lying in the brain, apparently about ¾ in. deep. The appearances are compatible with a calcifying abscess, hæmatoma, or new growth."

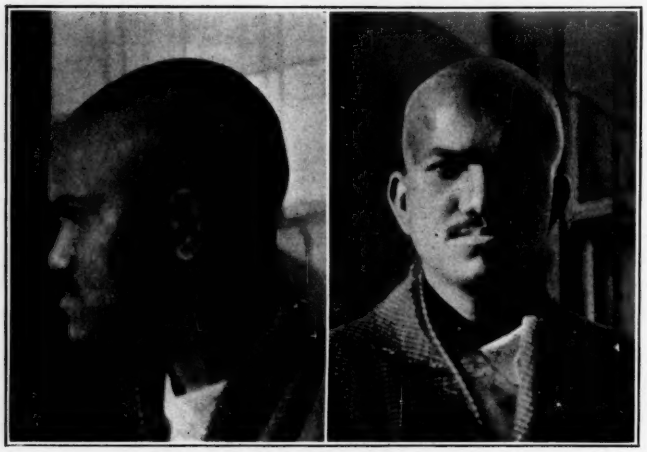

FIG. 129.—*Case* 1. The patient before operation.

PHYSICIAN'S REPORT.— "Heart, urine, chest, and lungs normal. The reflexes were normal. There was no Rombergism and there was no nystagmus."

FIG. 130.—*Case* 1. The patient after operation.

Personally I never saw this patient in any of his attacks, but when I saw him he looked dull and stupid (*Fig.* 129).

Judging from the reports of the radiologist and the ophthalmologist there was no doubt that there was a foreign body in his brain. There was also no doubt, judging from the history of attacks of headache and Jacksonian epilepsy increasing in frequency and severity, from the situation of the foreign body as shown by the skiagram, and from the eye symptoms, that they made a complete picture and made it reasonably certain that the foreign body (whether it was a calcified hæmatoma, or calcified tubercle, or a calcified abscess or a tumour) was the cause of all the man's trouble. I therefore resolved to operate with a view to removing the mass.

OPERATION.—On Saturday, August 29, I operated on the patient under

intrapharyngeal ether. Dr. Harte assisted me. I turned down a large scalp flap (shown in *Fig.* 130), taking as my base-line the top attachment of the ear. After denuding the skull bones of pericranium, I made four openings in the skull, with graduated burrs. Then with a hammer and chisel I removed the flap of bone between the four openings; the size of the flap is indicated in the X-ray photograph (*Fig.* 131).

Having removed this flap of bone, I left it lying on the scalp flap and attended to bleeding points. There was, strange to say, no bulging of the dura. So obvious was this fact that the onlookers noticed it. I then incised the dura in a cruciform manner. The brain did not bulge at all. I then insinuated my finger between the dura and the brain and orientated myself

FIG. 131.—*Case* 1. Skiagram showing the size of the bone flap. The skiagram is reversed.

in the middle fossa. My opening was in such a position as to make it easier for me to get my finger round the front of the temporal lobe than round the back. During the process of passing my finger round the back of the temporal lobe I felt a 'spiky' projection through the surface of the brain, and knew that I was on the foreign mass. I gently pushed my finger into the brain—the temporosphenoidal lobe—and gradually worked my way round the whole mass until I had it free in the brain. I then began to extract it, and with the assistance of the forefinger of my left hand brought it out from the opening in the skull. There was a good deal of oozing from the cavity in the brain, which I eventually stopped by repeated gentle plugging with gauze. Fearing a collection of blood in the cavity after the operation, I left a split rubber tube in the cavity, with a strip of gauze in it. The dura

was then folded back from the brain, but not stitched. The bone flap was replaced, and the end of the rubber tube brought through the posterior inferior 'trephine' opening and through a stab wound in the skin behind the ear. The scalp was then completely stitched up.

The whole operation lasted about an hour and a quarter to an hour and a half.

The patient was put to bed, and as his blood-pressure was fairly low, his temperature subnormal, and his pulse rapid, camphor, digalin, and pituitary were administered hypodermically, and salines subcutaneously and rectally.

The mass removed is shown in *Fig.* 132. It will be noticed how accurately this corresponds with the original skiagram taken before the operation.

Subsequent History.—There was some anxiety about the patient's condition for three weeks, but he looked well and felt well, and discussed how soon he would be able to get out of bed and use the commode and go to the lavatory. The rubber tube was removed after forty-eight hours. On Sept. 10 I did a lumbar puncture and had the fluid examined at the Institute for Medical Research. I received the following report:—

"Microscopic examination of the centrifugalized deposit of this fluid shows abundant polymorphonuclear leucocytes with some small lymphocytes (in the proportion of 4 : 1). No organism seen on direct examination. Cytological examination shows 2600 cells per cubic millimetre of the fluid. Cultivation has afforded a negative result."

Fig. 132.—The calcified mass removed in *Case* 1.

This suggests that blood had trickled down into the spinal column, probably through the cavity from which the mass was removed. On Sept. 13 his temperature and pulse were nearer normal. On the 14th, however, his temperature rose to 104° and his pulse-rate to 128. I suspected malaria, and treated him with quinine. His blood was not examined before giving him the quinine, but a day or two later gave a negative result. Examination of his stools was also negative.

We had in the meantime started saline bowel wash-outs on general principles on Sept. 10; this was continued till Sept. 23, by which time his temperature had settled, his stools were normal, and he was giving us no more anxiety. Before we arrived at this comfortable stage, however, we noticed that his mouth was very septic, and on two occasions we asked dentists to examine him and tell us whether in their opinion dental sepsis could be the cause of his temperature. One of them thought it might be, and the other thought it could not.

He was discharged from the nursing home on Oct. 4, and, as shown in *Fig.* 130, appears perfectly well. The photograph taken before operation (*Fig.* 129) is that of a sullen, morose, suffering individual; that taken after rather suggests a happy individual. The headaches have ceased, he has had no more attacks of Jacksonian epilepsy, and in my opinion the skull flap is fixed in position.

Pathological Report.—The specimen was examined at the South

African Institute for Medical Research (A. Sutherland Strachan), and the report was as follows :—

"The specimen was sawn through longitudinally, and portions taken for examination from the outer irregular surface and from the centre of the mass. Sections of these portions show a laminated calcified mass, with traces of altered blood pigment, and an amorphous mass with calcifying spicules scattered in its substance. There is no evidence of tumours or of tuberculosis. The features are those of a calcified hæmatoma." (*Fig.* 133.)

In view of the fact that the injury to the skull was on the right side, the hæmorrhage was undoubtedly due to *contre-coup* injury.

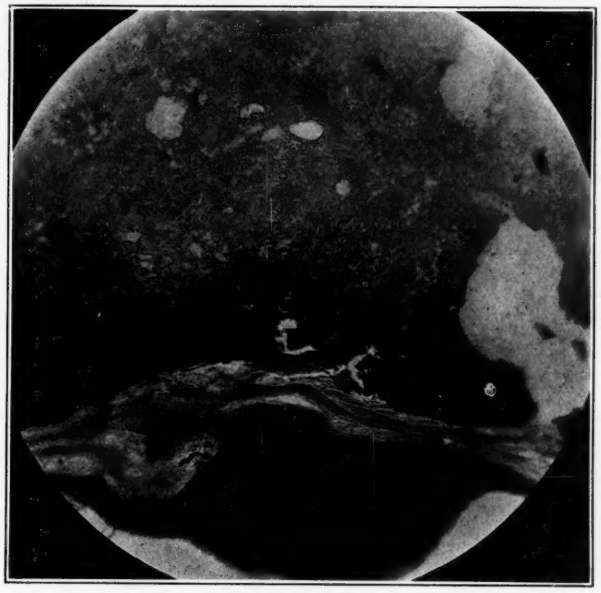

FIG. 133.—Microscopic section of the calcified specimen in *Case* 1.

Case 2.—A single woman, age 29, whose parents were a 'Malabar' coolie and a Hottentot. She came to hospital for chest trouble, cough, vomiting, and asthma, on Oct. 23, 1925. Her sinuses were suspected of being the focus of infection, and her skull was X-rayed, when an apparently calcified mass was discovered in the *right* cerebral hemisphere (*Fig.* 134). After this we obtained the following history, but without suggestion :—

HISTORY.—"Many years ago, when a child, she fell out of a tree and struck the ground with the *left* side of her head. She did not become unconscious. At the end of December, 1924, she had a headache in the frontal region, for which she took medicine. A few days later she noticed jerking movements of her *left upper limb*. Six months afterwards she had another attack of jerking movements of four hours' duration, but this time the left leg as well as the left arm were involved. A couple of months later another attack of headache superseded, followed by jerking movements of the left upper and lower limbs, and lasted about ten hours". She suffered from palpitation after the attacks.

One must assume that these jerking movements of her limbs are Jacksonian epilepsy. Since she has been in hospital, she has had one attack of headache, but no Jacksonian epilepsy.

On Examination.—The patient was a small coloured woman, with the left side of her body very slightly less developed than the right.

X-ray Appearances.—See Fig. 134.

Nervous System.—Reflexes: Cranial nerves, nothing abnormal. Left arm: biceps + + ; triceps + + ; brachioradialis + +. Right arm, normal. Left leg: knee-jerk + + ; ankle-jerk + +. Right leg: knee-jerk + ; ankle-jerk +. Abdominal reflexes elicited on either side. Plantar reflexes, doubtful. Right arm, muscle power good. Left arm, muscle power weak. Right leg, muscle power good. Left leg, muscle power weak.

Fig. 134.—*Case* 2. Skiagram showing an apparently calcified mass in the right cerebral hemisphere.

Sense of position with the right leg and arm, normal. Left leg, normal; left arm, impaired.

Sense of touch: right side, normal; left side, normal.

Sense of heat and cold: right side, normal; left side, cannot differentiate between heat and cold on the inner aspect of the tibia from knee to ankle.

Circulatory System.—Cardiac, nil.

Respiratory System.—Bronchitis.

Ophthalmologist's Examination.—Pupils equal and normal. Field of vision full. Discs normal.

Aural Surgeon's Examination—Nasal examination, negative; naso-

pharynx, negative; tonsils, small, flat, healthy. Some bad teeth. Larynx and epiglottis, vocal cords move freely. Hearing, whisper heard easily 6 ft. or more on either side.

L.M.T.: results of chronic suppurative otitis media, perforation in postero-superior quadrant, showing incus. R.M.T.: results of chronic suppurative otitis media, large postero-superior marginal scar.

Caloric test: left ear, nystagmus 50 sec., right; right ear, nystagmus 50 sec., left; past-pointing, not reliable.

Turning to left, 22 sec. after nystagmus. Turning to left, past-pointed 4 in. to left with the left, and 1 in. to the left with the right. Turning to right, 20 sec. after nystagmus. Turning to right, past-pointed 3 in. to right with the right, and 1 in. to right with the left.

Conclusion: Labyrinth tests yield normal results, bearing in mind that the left side of the body is weaker and not so well developed as the right.

DIAGNOSIS.—One is of opinion that there can be little doubt, judging from the history, the skiagrams, and the examination, that this woman's symptoms are due to the presence of a small 'foreign' mass in her brain, and that the mass is situated in the Rolandic fissure area on the right side of the brain near the arm area. The nature of the mass I believe is similar to that of the first case, namely, a calcified hæmatoma. In this case, however, unless the headaches and Jacksonian epilepsy become more frequent and severe, one feels that there is no justification for attempting the removal of the mass.

Of the fourteen references, I have been able personally to consult four; the others are not available in South Africa.

I am indebted to Dr. A. Sutherland Strachan, of the South African Institute for Medical Research, for the pathological report; to Mr. D. M. Greig (Edinburgh) for assistance in the matter of references; and to Drs. Harte, Campbell, and E. B. Israel for valuable assistance with the cases.

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MYELOMA AND CAVITIES IN BONE.

By R. P. ROWLANDS, London.

Myeloma is an innocent tumour of bone which grows from the red marrow, which it closely simulates in structure, being chiefly composed of multinucleated giant cells embedded in spindle and round cells. It has almost the same distribution as the red marrow, but is especially common in the upper end of the tibia and the lower end of the radius; the femur and the humerus are also frequently affected. It is curious, as Sir John Bland-Sutton[1] states, that this disease rarely affects the abundant cancellous tissues of the bodies of the vertebræ. There is a specimen in the Museum of the Royal College of Surgeons showing this growth affecting the patella; the limb was amputated after exploration.

Fig. 135.—*Case* 3. Myeloma of femur. Before operation.

It was, at one time, common to mistake this growth for myeloid sarcoma, but it is now generally recognized that myeloma is not malignant; it does not disseminate, but may be very destructive locally, if neglected. Under these circumstances the femur or the tibia may be so extensively involved that an amputation may seem to be the only reasonable and hopeful treatment. I am describing two cases to demonstrate that this view is fallacious and that the limb can be saved with complete restoration of function. I will also deal very briefly with some earlier cases which led me to what I regard as the correct treatment of this disease.

Case 1.—About twenty-five years ago a young Mauritian was hit on the outer side of the tibia by a cricket ball. Before long the tibia began to swell and cause severe pain. The swelling gradually increased, and when I saw him, some months later, there was a large swelling just below the knee, with egg-shell crackling over the outer tuberosity of the tibia, and undue mobility of the head of the fibula, which had been displaced outwards by the growth. At an exploration with the aid of a tourniquet, a large typical myeloma was found occupying about three-fourths of the upper

end of the tibia, some healthy bone remaining on the postero-internal surface. The growth had protruded out of its shell on the outer side, invading the extensor muscles, but had not pierced the articular cartilage into the knee-joint. It shelled out quite easily, and the walls of the cavity were carefully curetted with sharp spoons of various sizes. The cavity was washed out with an antiseptic solution.

Section proved that the growth was a myeloma, and the late Mr. L. A. Dunn agreed with me as to the diagnosis and treatment. Both he and I hoped that the limb could be saved, and that the cavity, which was temporarily drained, would gradually fill with bone. A Parisian surgeon, however, was sent for by the patient's father, and he urged amputation. The father agreed with him, and unfortunately I very reluctantly amputated through the lower third of the thigh. A naked-eye examination of the tibia after amputation, and eighteen microscopic sections of the wall of the cavity, showed that there was no sign of growth; therefore it is clear that, with more knowledge and confidence, the limb could have been saved and its functions restored. I am glad to say the patient was quite well when I saw him nearly twenty years later, when he came over to give what service he could during the War.

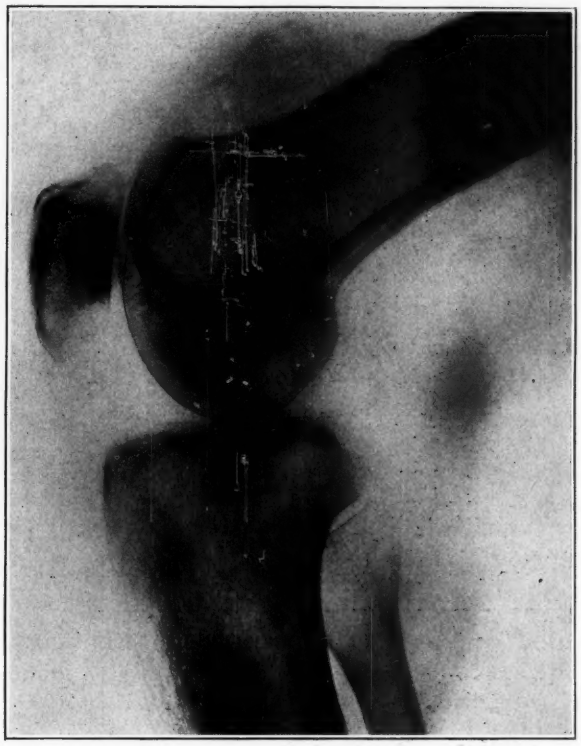

FIG. 136.—*Case* 3. Myeloma of femur. Before operation.

Case 2.—In May, 1914, the chauffeur to Dr. Duncan, of Lee Green, a man of 39, fell with his right knee doubled under him. He was in bed for two or three days, suffering considerable pain and swelling of the knee. I saw the patient in September, 1915, when a radiogram taken elsewhere had shown absorption of the centre of the bone. A diagnosis of central sarcoma had been made and amputation recommended, but the patient preferred to come to Guy's Hospital for treatment. I operated on Sept. 7. After applying a tourniquet around the root of the thigh, I made a longitudinal incision over the prominence on the front and inner side of the lower end of the femur, and removed a strip an inch wide of the shell covering the growth. This shell was thicker than I had expected, being about one-third of an inch. The growth was very soft; in parts it was like dark clot, and in others white and almost caseous. There was also in parts a greenish membrane between the growth and the shell of bone.

Dr. G. W. Nicholson immediately examined the growth, and found many giant cells; he reported that it was a myeloma and not a myeloid sarcoma. Therefore I scraped it out with great care and applied a solution of formalin (1–250) to the walls of the cavity and the lips of the wound, with the idea of destroying any cells of growth that might be left. The resulting cavity was very large, occupying the whole of the lower end of the femur and about three inches of the central canal of the shaft, which had been somewhat expanded. I scraped away until I reached the healthy marrow of the shaft.

After the operation the femur did not bend on exerting lateral pressure, nor did

it give way when one pushed the heel forcibly upwards. The wound was completely closed with the idea of letting the cavity fill with blood, which in time might be expected to ossify. If this plan should fail, bismuth paste or sterilized paraffin could be employed to fill the cavity, or a silver tube could be left in to drain it. When a large and extensive dressing had been applied and firmly bandaged, the tourniquet was removed and the limb well raised on pillows in bed.

Three weeks later there was a discharge of blood-stained serum from the wound, but the patient did well, had no fever and no further discharge. He wore a caliper splint for some months, and radiograms were taken from time to time showing the gradual ossification of the contents of the large cavity in the lower end of the femur. Ultimately the man made a complete recovery and discarded his splint. Unfortunately he was lost sight of during the War and has not been traced since.

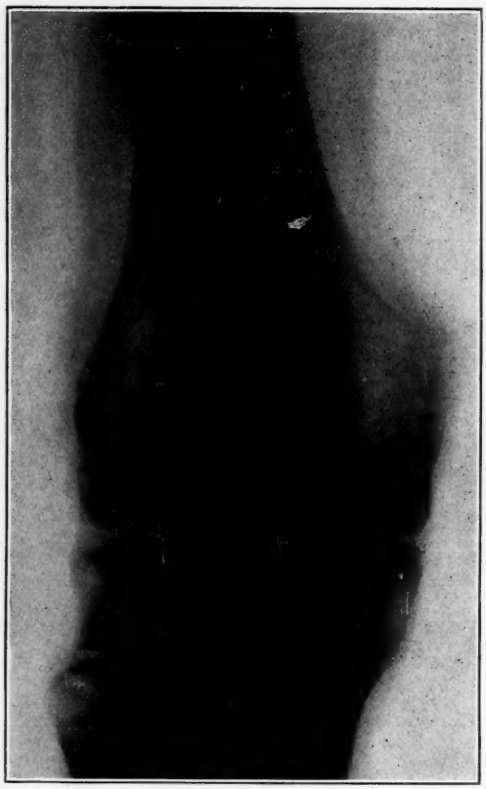

FIG. 137.—*Case* 3. Myeloma of femur. Seven months after operation.

Case 3.—E. V. B., a man, age 29, was admitted to Guy's Hospital on July 6, 1925. Eight months earlier, while playing football, he had noticed something 'go snap' on the inner side of the right knee. Two months later there was slight swelling of the right knee-joint, associated with pain. This swelling gradually increased, and in March, 1925, it had spread to the front and outer side of the thigh just above the knee. The knee was treated for synovitis and put in plaster for a month.

ON ADMISSION.—The swelling was then larger, firmer, and more adherent to the skin: there was no pulsation or eggshell crackling. Radiograms (*Figs.* 135 and 136) showed a large, circular, pale area in the region of the external condyle and shaft of the femur. They indicated a central tumour which had pierced the bone in front and had stripped the periosteum considerably; the line of this showed ossification.

OPERATION.—The femur was explored through an incision along its outer border, after applying a tourniquet around the upper third of the thigh. As anticipated from the roundness and sharp margin of the pale area in the femur, the growth turned out to be a myeloma. It shelled out easily, and the surrounding parts were thoroughly scraped; but the shell had been perforated by the growth posteriorly, towards the popliteal space, and also on its antero-external surface. The tumour was the size of a tennis ball, partly inside and partly outside the femur. Microscopic section proved it to be a typical myeloma. The cavity was allowed to fill with blood, and later to ossify.

A fortnight after the operation the patient walked with a caliper splint, which he was allowed to leave off at night after four months, and by day after five months, when he was able to stand and walk quite well. Several radiograms, taken at intervals, had shown increasing ossification of the contents of the cavity, and the limb had become more and more firm. He is now able to bend the knee to a right angle, and to walk quite well without support or stick. Radiograms taken early in 1926 show no sign of recurrence of the disease (*Figs.* 137 and 138).

Case 4.—A. G., a man, age 20, was admitted to Guy's Hospital on Aug. 24, 1925, for X-ray examination of the left knee. He had first experienced pain in the knee when walking, two months before. This had passed off for a week, but had recurred with increasing intensity and had not yielded to palliative treatment. The X-ray plate showed a large circular pale area in the internal tibial tuberosity suggestive of myeloma. This area was very tender to touch and painful on movement. An operation was performed four days later. On exposing the periosteum on the inner side and incising it, a brown coloured 'jam-like' looking mass was seen to lie immediately underneath. The cavity containing this pulp was scraped out with a sharp spoon, and two samples of the contents were taken for microscopic examination. The cavity measured about two inches in diameter, and was egg-shaped. A strong

FIG. 138.—*Case* 3. Myeloma of femur. Seven months after operation.

solution of biniodide of mercury was used to kill any cells that might remain. The periosteum was then sutured and the wound closed. Microscopic examination proved the growth to be a typical-celled myeloma.

The patient was fitted with a caliper which he wore for four months. His recovery was so complete that in February, 1926, when he was asked to show his scar, he uncovered the wrong leg.

The last three cases show conclusively that the best way of treating myeloma of bone when it is imperative to prevent shortening, as is usually the case with the lower limb, is by very carefully shelling out the growth. They also show that the best way of treating the resulting bone cavity is by closing the wound completely and letting the cavity fill with blood; spontaneous

ossification usually takes place, with complete restoration of function. It is, of course, essential to prevent spontaneous fracture and to maintain the length of the limb and enable the patient to walk about; this can be done by means of a caliper splint, which is used for about six months after the operation.

Case 5.—R. W., a little boy, now 8 years old, was operated on for me by Mr. W. H Ogilvie five years ago, when he was my surgical registrar. He has shown and discussed this case before the Royal Society of Medicine.[2] It was a case of osteitis fibrosa of nearly the whole of the shaft of the right humerus (*Fig.* 139).

FIG. 139.—*Case* 5. Fibro-cystic disease of the right humerus. Before operation. Practically the whole of the shaft was excised.

After a preliminary exploration and microscopic examination establishing the diagnosis, Mr. Ogilvie removed nearly the whole of the shaft of the humerus subperiosteally. The immediate insertion of an autogenous bone-graft, which had been contemplated, had to be abandoned because the little boy was too exhausted for further prolongation of the operation. This proved to be very fortunate, for it led us to an important discovery, namely, that the graft was unnecessary, for the cavity in the arm filled at first with blood and then very rapidly with bone.

Two months later the boy was able to use his right arm freely and to raise his right hand to the normal extent above his head. He made a rapid and complete recovery. He was radiographed in February, 1926, by Dr. J. M. Redding (*Fig.* 140), who reported: "Complete ossification of the humerus. Comparatively slight deformity; slight shortening. No evidence of recurrence of disease". Even the central canal of the humerus had been restored.

The result was, in fact, as good as, if not better than, that of a very successful case shown by Mr. Gordon Bryan[3] in which he had used a boiled beef-bone graft. If a graft is unnecessary, it is clearly better to do without it, for grafts may give rise to many troubles and some dangers: amongst these may be mentioned spontaneous fracture of the graft or of the parent bone, necrosis, sinus formation and imperfect union.

I have successfully used the same simple method for filling other large aseptic bone cavities, such as those resulting from simple cysts. Some infected cavities have been similarly treated with success, after taking pains to clean them before, or by excising their walls during, the operation. Infection of bone is, however, very difficult to eradicate.

Since the lecture of which this paper is an abstract was delivered at Guy's Hospital, on Feb. 11, 1926, an excellent paper by Major J. B. Hance, I.M.S., has appeared in the January number of the *Guy's Hospital Reports* which reached me on Feb. 24. Major Hance refers to Mr. Alan Todd's[4] brilliant case

reported in the *Proceedings of the Royal Society of Medicine* (Clinical Section). In these papers autogenous cancellous grafting of the cavities is strongly recommended. My experience proves that grafting is superfluous, although it may possibly secure more rapid ossification.

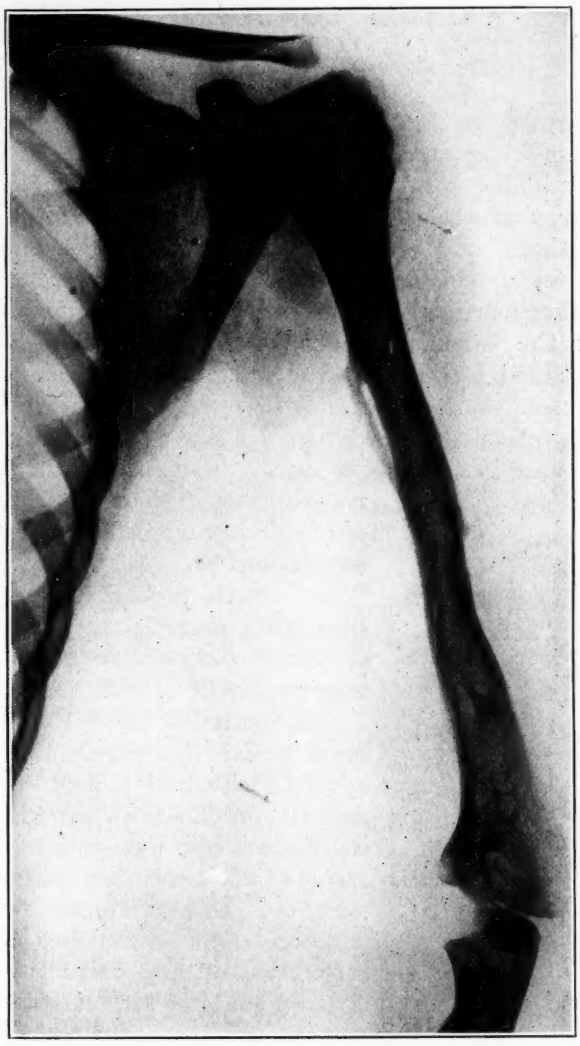

FIG. 140.—*Case* 5. Fibrocystic disease of right humerus. After operation. Note re-formation of the shaft of the humerus without bone-grafting.

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URINARY CALCULI COMPOSED OF BACTERIA.

By R. OGIER WARD, London.

Soft 'stones' formed in the urinary system are exceedingly rare. In some instances they are composed of fibrin; Gage and Beal[1] collected a series of these in 1908, and Thomson-Walker[2] describes a case. Such masses are soft, of greyish colour, and are composed of layers of fibrin with perhaps a few crystals among them, and one view of their nature is that they represent that organic material which is thought to cement together the crystallized portions of ordinary urinary calculi—or, in other words, that they are the mortar without the bricks; whilst another supposition is that they are formed from retained blood-clots, the fibrin of which has become moulded into regular masses. But the calculi here described belong to another and even rarer variety of soft 'stones', namely, those which are composed entirely of bacteria. J. and W. Israel[3] mention these in their recent book, giving references to four writers, and these are the only other cases of which I can learn. The concretions are described by these writers as "soft elastic bodies varying in size from a bean to a cherry; they are round or oval, or tetrahedraform in shape, and are composed entirely of coli bacteria".

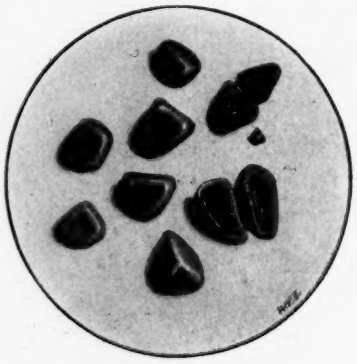

Fig. 141.—Some of the soft calculi passed.

A patient with this disease has recently been under my care. She was a female, age 35, who had suffered in the past from malaria, typhoid fever, and a tendency to diarrhœa. She was stated to have had many attacks of toxæmia and was exceedingly neurotic. Fifteen months previously she had a severe right renal colic, this being the first intimation of any urinary disorder; hæmaturia accompanied the colic and lasted for a month, the urine being usually claret-coloured. At the end of the month she had another severe colic on the same side, and this was followed by acute renal infection, the temperature often going above 104°. About this time she was X-rayed, and one calculus was discovered which was judged small enough to be passed. About a month later—that is, three months from the onset of symptoms—there occurred several milder attacks of colic during which some small soft 'stones' (*Fig.* 141) were passed. Subsequently, after a more severe attack, the calculus seen on X-ray examination was got rid of, together with three of the softer variety. Throughout the subsequent ten months there were many attacks of right renal colic and nearly a hundred soft 'stones' were passed.

A pyelogram carried out by Dr. Rolfe Kingsley in New York showed the right kidney to be dropped and the ureter kinked directly below the pelvis, which seemed enlarged. The opaque fluid was dispersed throughout the pelvis by the presence of the stones. Pyuria persisted, but for various reasons operation had to be postponed. Some months later, however, there was another acute attack of obstruction and inflammation of the right kidney. I removed the right kidney and the calculi which it contained. The urine shortly before this attack had much pus in it, a few red blood-cells, and some transitional epithelial cells, whilst on culture a pure and abundant growth of *Bacillus coli communis* was obtained. The patient recovered from the operation, and all pyuria ceased at once, although three weeks later the organisms could still be cultured.

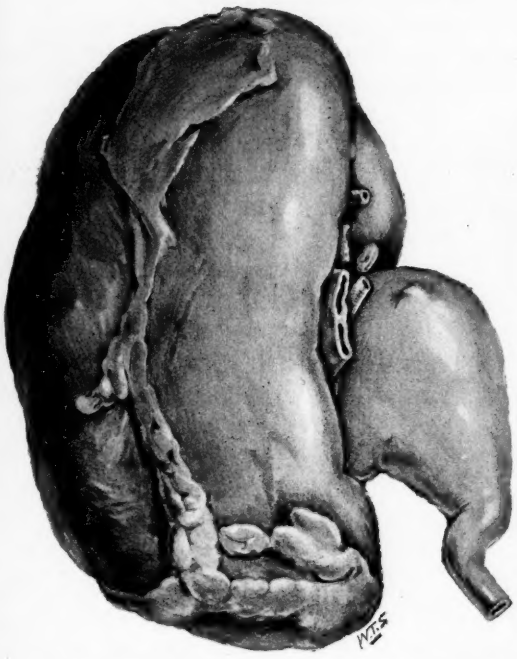

FIG. 142.—Showing the dilatation of the renal pelvis and the kinked ureter.

Fig. 142 shows that the renal pelvis is moderately dilated, and the kink of the ureter, which was demonstrated by pyelography, can also be seen.

Fig. 143 depicts the interior of the kidney after the highly purulent urine and most of the calculi—of which it contained over a hundred—had been removed. The extent of the hydronephrosis, and manner in which the concretions were distributed throughout the dilated calices, is well seen; whilst the report by Mr. G. L. Keynes which follows describes the microscopical appearances.

FIG. 143.—Interior of kidney after the removal of most of the calculi.

Section of one Pyramid and Corresponding Cortex shows: (1) Dilatation of all the tubules. (2) Hyaline degeneration of some of the glomeruli. (3) Infiltration of the interstitial tissue in

certain segments, from apex of pyramid to cortex, with large numbers of leucocytes; in the cortex the appearance suggests that small abscesses are beginning to form. (4) Some of the collecting tubules contain casts formed of leucocytes.

The microscopic appearances indicate: (1) Chronic nephritis; (2) Hydronephrosis; (3) An acute ascending infection of the whole kidney.

FIG. 144.—Magnified cross-section of one of the concretions.

Fig. 144, which is a magnified cross-section of one of the concretions stained by a silver process by Dr. F. W. Twort, shows its laminated structure, and that the external surface of each layer is most darkly coloured.

When this is compared with *Fig.* 145, which is a more highly magnified view of the darkly coloured outer part of one of the laminæ, it becomes obvious that the 'stones' are composed almost entirely of bacteria, which are more degenerate towards the internal parts of each lamella, and here more feebly take the basic aniline dyes.

Dr. H. E. Archer's report details the chemical analysis and correlates this with the microscopical appearances described by Dr. Twort:—

Small tetrahedraform faceted calculi. Colour varying between grey-brown and grey. Surface smooth. The majority have a superficial white coat of 'fibrinous' material. Some calculi appeared to have a minute white nucleus.

		For one calculus.
6 average calculi weighed	0·5135 grm. ..	0·0856 grm.
,, ,, ,, dried weight	0·1275 ,, ..	0·0212 ,,
,, ,, ,, ashed weight	0·0291 ,, ..	0·005 ,,

,, ,, ,, extracted with ether yielded a minute amount of lipoidal matter which gave a very definite reaction for cholesterol, probably derived from blood (which normally contains about 0·16 per cent).

Chemical examination of ash showed: calcium; phosphate; sodium and magnesium (trace); iron (trace); chloride (trace).

Prolonged peptic and tryptic digestion had practically no effect upon the organic matter of the calculus. This indicated that the material is probably not fibrin.

Conclusions.—Comparing the chemical findings with the microscopical sections, the central portion appears to be the remains of a minute blood-clot, and the outer layers to consist of bacteria and organic débris with a small amount of calcium and magnesium salts.

The cementing layers appear to be of a mucoid nature.

It remains only to make a few comments on the case. There was a history of bowel disorder which may have been the source of the infection

of the urinary tract; and the ureteropelvic kink and ptosis, by producing stasis in the right kidney, was probably the factor which determined its localization in that organ. The writer cannot, however, offer any explanation of the special conditions which obtained in the case, and which caused the bacteria to cohere and to form these very numerous concretions (for there were in all more than two hundred of them), whilst in other patients infected with the same organism only a bacilluria or a urinary infection results. Presumably the mucinous cement material is the factor essential for cohesion. Nor is it certain whether the 'stones' were all formed at one time or were being continually produced, though the laminated appearance favours the latter suggestion.

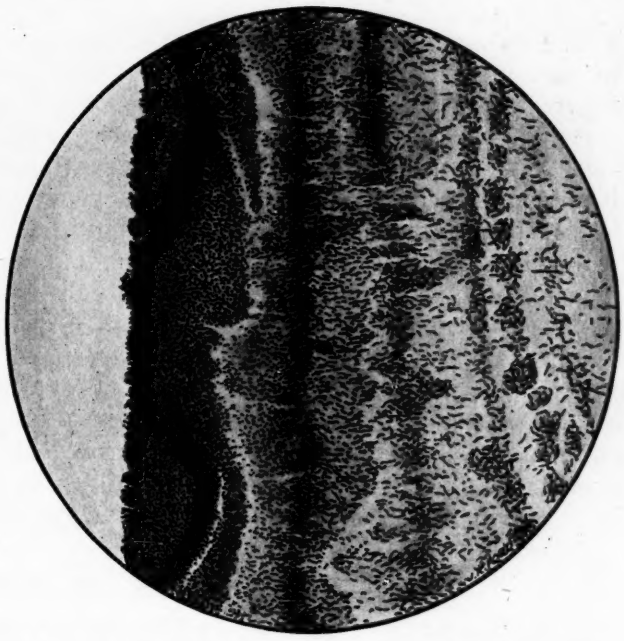

FIG. 145.—More highly magnified view of outer part of one of the laminæ.

The diagnosis of such a case is unlikely to present any special difficulties, for the 'stones' are so soft and elastic that some are sure to be passed, when a careful examination will reveal their nature. A radiogram by Dr. N. S. Finzi, using the Potter-Bucky diaphragm, showed their outline shadows very faintly, but a pyelogram will probably always be necessary to make this obvious.

As to treatment, in the case here described nephrectomy was essential because a pyonephrosis was forming, but in other instances it might be advisable to postpone this if there were reason to think that the calculi were being discharged from the kidney and others were not forming, for their soft elastic nature renders their passage along the ureter much more easily effected than is the case with the common varieties of calculi.

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TUBERCULOSIS OF THE MALE BREAST.

By R. H. JOCELYN SWAN and H. J. B. FRY, London.

Tuberculosis of the male breast must be accounted a pathological curiosity, for not more than eleven cases have been recorded in the literature since tuberculosis of the breast was first described nearly a century ago.* The present forms the twelfth of the published cases, several of which, however, cannot be regarded as definite from a pathological standpoint. Indeed, it is a remarkable fact that, in spite of the widespread prevalence of tuberculosis in its various forms, tuberculous infection of the breast, even in the female, is a rare condition. Fewer than two hundred cases have been reported in all, including the male cases. In published collections of tumours of the breast they form not more than 0·5 to 1 per cent. The records of the Cancer Hospital for the last quarter of a century have been searched; and in this series not previously reported, out of 2542 tumours of the breast of all kinds, there have been 10 cases of tuberculosis of the breast, including the male case reported here, all of which have been verified by microscopical examination. This gives a percentage of 0·4 for this series, in which the only male case during this period of twenty-five years is the present one.

CLINICAL NOTES OF THE CASE.

F. P., age 42, chauffeur. Admitted May 20, 1924, under Mr. Jocelyn Swan.

History.—The patient complained of a swelling in the left breast which he had noticed for three months. This had become gradually larger, and during the last month there had commenced some retraction of the nipple. The swelling was not painful, but pressure on it caused discomfort. In 1900, twenty-four years previously, the patient was operated upon for tuberculosis of the right hip-joint, which was ankylosed. There was no history of other illness.

On Admission.—The patient appeared healthy, and was well nourished. There was a rounded, hard, somewhat nodular swelling in the region of the left breast about two inches in diameter; it was fixed to the subjacent pectoral muscle and the nipple was slightly retracted; the skin of the areola and of the part immediately surrounding the latter was thickened and adherent to the mass, but there was neither redness nor œdema. Several small glands were palpable in the left axilla. A diagnosis of carcinoma of the breast was made, itself a somewhat rare condition in the male.

Operation.—Radical removal of the breast was advised and carried out on May 24, 1924. An incision was made from the left side of the umbilicus

* Since the above was written one more case has recently been described by Turco.[15]

obliquely upwards across the costal margin and the pectoral region internal to the anterior fold of the axilla and crossing the anterior fibres of the deltoid muscle, joined in the central part by another incision which enclosed an elliptical area of skin around the tumour in the breast. Skin and fascial flaps were turned back, and the whole breast was removed, together with the sterno-costal portion of the pectoralis major muscle, the pectoralis minor, the upper aponeurosis of the rectus muscle, and the fascia covering the serratus magnus and the contents of the axilla, the axillary vein being cleaned as high as the costocoracoid membrane. The wound healed without trouble and the patient made a complete recovery.

Pathological Examination.—When examined in the laboratory the breast was found to contain an abscess cavity immediately beneath the nipple in the fatty areolar tissue between the pectoralis major and the skin. The cavity was irregular in shape, lined by a thick wall of yellowish caseous granulation tissue, and limited by an outer layer of hyaline fibrous tissue (*Fig.* 146). A small quantity of rather thick yellow pus was evacuated on opening the cavity. On examination this pus showed a considerable number of acid-fast bacilli, morphologically typical tubercle bacilli. No other organisms were found.

Fig. 146.—Tuberculous abscess in the male breast.

Histological examination of the breast tissue showed an abscess cavity limited by a thick wall of fibrous tissue penetrated by chronic inflammatory cells (*Fig.* 147). The cavity was lined by granulation tissue, and contained acute and chronic inflammatory cells, connective-tissue cells, and fibroblasts in various stages of fatty degeneration and disintegration. Numerous plasma cells were present, with many polymorphonuclear cells and patches of red cells. Small giant cells were present in the wall of the abscess (*Fig.* 148). No fat necrosis was evident either in the abscess or in the neighbouring breast tissue. An adjacent lymph gland showed no tubercles, and, in fact, no marked pathological changes. In view of the positive character of the bacilli in the pus and of the histological appearances, animal inoculation was not performed.

Subsequent to this pathological finding, the patient was subjected to a further critical examination to discover other evidence of tuberculous disease. Mention has already been made of the previous tuberculous hip-joint, ankylosed after an operation twenty-four years previously, but no evidence was obtained of any pulmonary infection and no tubercle bacilli could be found in the sputum. Small hard nodules were found in the epididymis on both sides, and one was also found in the upper part of the right lobe of the prostate. In the absence of any history of venereal disease, these were looked upon as old foci of tuberculous infection, but no other suspicious lesions could be found.

FIG. 147.—Photomicrograph of wall of tuberculous abscess. (× 20.)

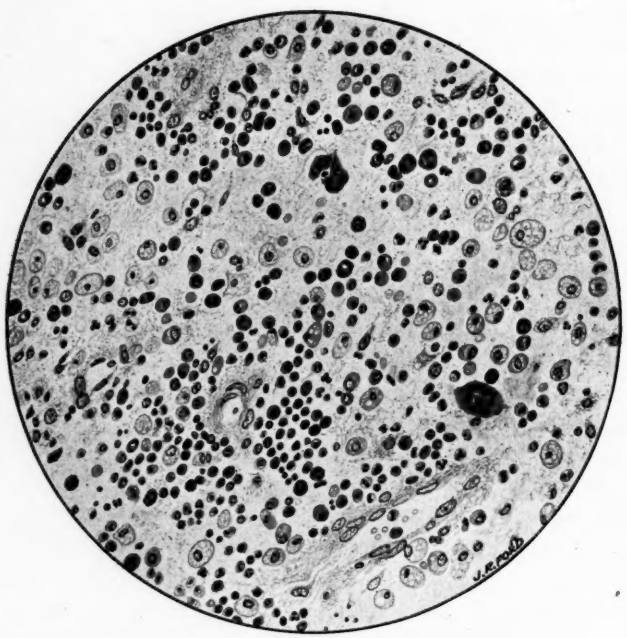

FIG. 148.—Photomicrograph of tuberculous abscess showing cellular contents. (× 135.)

HISTORICAL.

Tuberculosis of the breast was first described by Sir Astley Cooper[1] in 1829, the earliest case in the male being reported by Heyfelder[2] in 1851. No microscopical or bacteriological proof is available in this case, so that its nature remains open to doubt, and it was therefore rejected by Scudder.[3] From the clinical description, however, it seems probable that Heyfelder's case was tuberculous in nature. The first case in the male in which tubercle bacilli were demonstrated seems to be the one by Schede[4] in 1893. Other cases in the male have been reported by Poirier,[5] Demme,[6] Delbet,[7] Ferguson,[8] Hebb,[9] Parsons,[10] Khesin,[11] Ressiguie,[12] and the last, in 1916, by Durante and MacCarty.[13] Of these only seven have been definitely proved by satisfactory pathological and bacteriological findings, viz., those of Poirier, Schede, Hebb, Demme, Khesin, Ressiguie, and Durante and MacCarty. The case quoted by Delbet was an oral communication by Thierry, without any published details, and is therefore a matter of conjecture only. The cases of Schede and Ferguson are practically devoid of details.

ANALYSIS OF CASES.

Incidence.—The remarkably small number of cases described in the male, ranging over a period of nearly one hundred years since the condition was first described, clearly indicates the rarity of this infection. It forms, indeed, less than 0·05 per cent of all breast cases. In the Cancer Hospital series the percentage is 0·04, which accords well with the proportion of published male and female cases. This is of some interest when considered in relation to the general incidence of tuberculosis in other forms in the male and calls for some explanation of its pathology.

Predisposing Causes.—

1. *Age.*—The common period appears to be the third and fourth decades of life, and all but five of the cases can be included in this period, a remarkable exception being the case described by Demme[6] of direct infection in an infant of 6 months. The oldest case recorded is 52.

2. *Heredity.*—Evidence of an hereditary susceptibility has been found in only one of the cases recorded (Demme).

3. *Antecedent Injury or Disease.*—In two instances there was a history of injury followed by the tumour, and in another a previous abscess, but in the majority of cases this does not appear to influence the condition.

Classification.—Tuberculosis of the breast has been divided into two groups, primary and secondary. The former comprises the cases in which there is not elsewhere a demonstrable tuberculous infection from which the lesion of the breast can be derived. Infection is, therefore, considered to be primary when it occurs either directly through the skin of the breast or through the ducts of the nipple, or if conveyed to the breast through the blood-stream from a remote portal of entry. In this series only four cases can be considered to be primary, in one alone of which is there evidence of direct infection (Demme). In two cases the evidence is insufficient as to the mode of infection. In six cases the infection of the breast was secondary to foci of the disease elsewhere. This is evidently the commonest form. In two cases

there was a history of joint tuberculosis (Khesin, Swan and Fry); two were due to osteitis of the ribs (Parsons, Ferguson); one to tuberculous lymphatic glands (Heyfelder); and one to pulmonary tuberculosis (Durante and MacCarty). The channels of infection were, therefore: (1) By the blood-stream; (2) By direct extension; (3) By lymphatic channels.

Types.—Various pathological types of tuberculous disease of the breast are described, namely: (1) Acute miliary tuberculous mastitis; (2) Nodular type; (3) Sclerosing type; (4) Mastitis obliterans.

In the male, only the nodular variety appears to have any place, especially the form of the intraglandular cold abscess of Roux, which is derived by a process of caseation and liquefaction from the nodular variety. In five of the published cases there was formation of abscesses of varying size. In Heyfelder's case the abscess extended from the acromion process to the lower border of the nipple; in others it was of small size. In the case described here the typical glandular cold abscess is shown in *Fig.* 146. The nodular type was present in five other cases in which a small lump was felt in the breast.

Bacteriology.—The condition can only be recognized with certainty by the finding of tubercle bacilli in the tissues or in the pus of the abscesses, by cultivation from pus, or by animal inoculation in suspected cases. In only three cases out of the twelve has this condition been fulfilled (Schede, Demme, Swan and Fry). The practical difficulty of finding scanty tubercle bacilli in tissues may account for this. In the case described here tubercle bacilli were numerous in the pus of the abscess. In only one of the cases has animal inoculation been practised.

Microscopic Anatomy.—In six out of the twelve cases the diagnosis rests upon microscopical appearances apart from the finding of tubercle bacilli. In

SUMMARY OF REPORTED CASES O

Reporter	HEYFELDER	POIRIER	DEMME	DELBET	HERB	SCHED
Year ..	1851	1882	1889	1892	1893	1893
Age ..	26	46	6 months	—	31	42
Occupation	Shoemaker	Road labourer	—	—	Gas-stoker	—
Heredity ..	—	Nil	Active pulmonary tuberculosis in mother and sister	—	Negative	—
Antecedent Condition of Breast	—	Blow in this region 2 years previously	—	—	Injury 8 months previously	—
Type ..	Secondary	Primary	Primary	—	Primary	—
Initial Symptoms	Lump	Two vesicular nodules; indurated bases	Swelling and abscess	—	Lump following injury	Absces —

most of these typical tubercles were found, with a greater or lesser degree of fibrosis, caseation, and necrosis, together with giant cells and chronic inflammatory cells. It must be remembered, however, that the condition of fat necrosis in the breast first described by Lee and Adair[14] may give rise to somewhat similar appearances, and care must be taken to distinguish the latter from tuberculous disease. Possibly some of the cases recorded may have been due to fat necrosis.

Symptoms.—The course of the disease is rapid in the male—not more than three to six months, and shorter than in the female. This is evidently due to the greater ease with which a small lump is recognized in the rudimentary male breast, and treatment more rapidly instituted.

Early symptoms: In at least half of the cases the initial symptom was the discovery of a lump in the breast. In only two cases was there pain connected with it. *Side*: the distribution between the two sides is about equal, four being in the right breast and five in the left, the remainder not being recorded. In the female the right is more often affected than the left side. The *site* is usually in the neighbourhood of the nipple—this was the position in four cases; in the lower quadrant, one; outer and inner sides, one each. In the female it is more generally the upper and outer quadrant which is affected. The skin was adherent in four of the cases where this was noted, and showed some inflammation in four. The nipple was retracted in two of the cases in which an abscess lay beneath it, but there was no discharge from the nipple in any. Axillary glands were enlarged in two of the cases. Sinuses were present in only one of the cases (Demme). The general condition of the patient appears to have been good in most instances, though the physique was poor in two.

TUBERCULOSIS OF THE MALE BREAST.

Ferguson	Parsons	Khesin	Ressiguie	Durante and MacCarty	Swan and Fry	Turco
1898	1907	1909	1909	1916	1925	1925
—	38	23	40	52	42	23
—	Labourer	—	—	—	Chauffeur	Warehouseman
—	—	—	—	—	Negative	Negative
—	—	—	Abscess 2 years previously	—	Healthy	Anti-typhoid inoculations
Secondary from rib	Secondary	Secondary	Primary	Secondary	Secondary	Primary
—	Lump	Lump	Lump	Abscess	Lump	Lump

Continued on next page

SUMMARY OF REPORTED CASES OF

Reporter	HEYFELDER	POIRIER	DEMME	DELBET	HEBB	SCHEDE
Location ..	Above nipple	3 cm. outside nipple	—	—	Inner side	—
Duration ..	2 months	3 months	From age of 4 days	—	6 months	
Size ..	Acromion to right border of sternum, below to the nipple	6 × 4 cm., oval	2·5 cm.	—	Small hen's egg	—
Side ..	Right	Right	Left	—	Left	—
Pain ..	—	Present 3 weeks	—	—	None	—
Attachment to Skin	Not adherent	Adherent; not to pectoralis major	Adherent	—	Not adherent to skin; slightly to muscle	—
Sinuses ..	—	No	Present	—	—	—
Nipple Retraction	—	No, but thickened	—	—	—	—
Axillary Glands	—	Affected	Affected	—	—	—
General Health	Poor physique	Good	Poor	—	—	—
General Examination	Swollen glands in neck with scars	No pulmonary or other signs of tuberculosis	—	—	—	—
Treatment	Incision 2in. and drainage	Excision, with axillary glands; muscle left	Ulcer scraped; iodoform dressing	—	Excision with removal of muscle	—
Result ..	Complete recovery	Uninterrupted recovery	Recovery	—	—	—
Pathology ..	—	Tubercle	Tubercle	—	Tubercle	—
T.B. found	—	—	Yes	—	—	Yes
Animal Inoculation	—	—	Positive	—	—	—

TUBERCULOSIS OF THE MALE BREAST—*continued.*

FERGUSON	PARSONS	KHESIN	RESSIGUIE	DURANTE AND MACCARTY	SWAN AND FRY	TURCO
—	Under nipple	—	Near nipple	Lower quadrant	Under nipple	Upper inner quadrant
—	3 months	—	3 months	—	3 months	3 years
—	$4 \times 5 \times 1\frac{1}{2}$ in.	—	Horse-chestnut	—	2 in. diameter	Half a hen's egg
—	Left	Right	Right	Left	Left	Left
—	Present	Present	At times	—	None	Present
—	Abscess adherent to superficial and deep structures	—	—	Slight inflammation	Adherent to skin and muscle	Lightly adherent to skin and to deep structures. Skin never reddened
—	—	—	—	—	Absent	None
—	Yes	—	—	—	Yes	Yes
—	—	Affected	—	—	Affected	Affected
—	—	—	Good	Poor	Good	Poor
Tuberculosis of rib	—	Antecedent joint tuberculosis	Negative	Pulmonary tuberculosis, empyema left side	Ankylosed tuberculous right hip; tuberculous nodules in epididymes and right lobe of prostate	No other evidence of tuberculosis
Extirpated while dealing with rib	Amputation of breast and part of pectoralis major; rib scraped	Amputation and clearing of axilla	Amputation with removal of pectoralis major	—	Radical excision	Amputation with partial removal of pectoral muscles and clearing of axilla
—	Uninterrupted recovery	—	—	—	Good	Excellent
—	Caseating tuberculous material (naked eye)	Tubercle	Tubercle	Tubercle	Abscess cavity; giant-cell systems	Abscess cavities; giant-cell systems
—	—	—	—	—	Yes	—
—	—	—	—	—	—	—

Differential Diagnosis.—This lies between the following conditions:—

1. *Ordinary Pyogenic Mastitis and Abscess.*—The greater chronicity of the tuberculous condition, the freedom from pain, and the absence of acute inflammation afford some indication. The pyogenic cases can probably be settled by bacteriological methods.

2. *Granulomata.*—Actinomycosis of the breast is rare and tends to the formation of sinuses, in the pus from which the ray-fungus can be found. Syphilitic conditions, such as gummata of the breast, are equally rare, and probably could not be distinguished except by a positive Wassermann reaction and by microscopical examination.

3. *Tumours,* whether simple or malignant, such as the fibro-adenomata or carcinomata of the male breast, are rare, but cannot easily be distinguished from a tuberculous infection except by microscopical methods.

4. *Fat necrosis* is also a condition which closely simulates both tuberculous and malignant disease (Lee and Adair[14]), and would require microscopical diagnosis.

Prognosis.—This appears to be uniformly good. In all cases recovery after surgical treatment was satisfactory.

Treatment.—Radical excision of the breast seems to be the method chiefly practised, together with removal of all tissues involved, with or without clearing of the axilla. Incision and drainage has also proved effective. Treatment, no doubt, would depend on correct diagnosis, which is not easy to determine prior to operative measures.

CONCLUSIONS.

Tuberculosis of the breast in the male is a very rare condition. In consequence of its rarity and from its nature it is difficult to diagnose and may easily be mistaken for carcinoma. Its rarity is probably due to the small size, rudimentary character, and absence of functional activity in the male breast, with its concomitant lack of vascular and lymphatic circulation.

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SPRENGEL'S DEFORMITY WITH PARAPLEGIA.

By MACDONALD CRITCHLEY, London.

Congenital elevation of the scapula, as described by Eulenburg, and later by Sprengel, is by no means common. The present case is of particular interest in that the subject is an adult, and has been under observation for sixteen years. The association of the scapular deformity with other bony defects in the skeleton is of interest—though not rare. The co-existence of a paraplegia with Sprengel's deformity, however, renders the case unique. The causation of the paraplegia is not clear, and a discussion as to the pathogenesis entails speculations of great importance.

Fig. 150.—Photograph illustrating the left-sided hemiatrophy and scoliosis; the tilted attitude of the head is well shown. The contour of the spinous processes and of the left scapula have been marked out with ink.

DESCRIPTION OF THE CASE.

F. L., age 45, a carman, was admitted to the National Hospital, Queen Square, under the care of Dr. James Collier. His chief complaint was of stiffness in the left leg and difficulty in walking. He stated that ever since the age of 2 years a deformity of the left shoulder and neck had been present, so that his left arm was weak and could not be raised above his head. Except for occasional darting pains in the shoulders and limbs, he remained well until the age of 30, when, in a drunken brawl, his arms were twisted behind his back. He felt a severe pain in the thorax, and both legs became numb and useless. The weakness in the legs had persisted ever since, accompanied by numbness and dull aching in the right leg. About three years before admission he began to have feelings of stiffness in the left leg, and aching pains in the left chest. No sphincter troubles

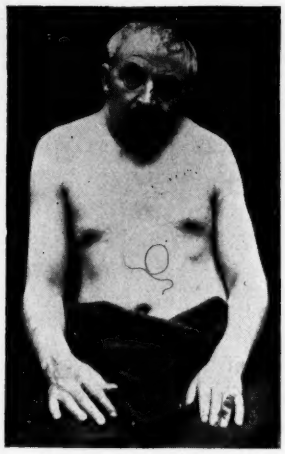


Fig. 149.—Front view of patient. Note the drooping of the affected shoulder. The facial asymmetry is quite apparent.

had ever been present. Apart from an attack of scarlet fever, and also of gonorrhœa at the age of 20, his previous health had been good. Syphilis was denied.

His parents were healthy and his six brothers and one sister were alive and well. The patient was a single man and had no children. There was no history obtainable of any similar deformity amongst the other members of the family.

Physical Examination.—The patient was a man about 5 ft. 2 in. in height. His build was remarkable in that he appeared to have scarcely any neck at all. The left side of the head was tilted down towards the shoulder. The interval between the right side of the occiput and the back of the trunk was occupied by a prominence which was felt to consist of scoliotic cervical vertebræ. The right shoulder blade lay at a higher level than the left, and could be felt to be elevated and rotated upwards, so that the vertebral border lay under the trapezius. There was a very sharp angular scoliosis of the cervical and upper thoracic vertebræ, the apex of the gibus being to the right. (*Figs.* 149–152.) The angles of the ribs were somewhat more prominent on the right side, whilst the left half of the chest bulged in front. The left arm and leg were seen to be a little smaller in girth than the right, though the left leg was a little longer.

DIMENSIONS.

	Right.	Left.
Circumference of upper arm	10½ in.	10½ in.
Length of forearms	10 „	9½ „
Circumference of forearms	10 „	9½ „
Circumference of hand (around knuckles) ..	8 „	7¼ „
Length of legs (from anterior superior iliac spine to tip of external malleolus) ..	33 „	34¾ „
Circumference of thighs	18 „	19½ „
Circumference of calf	13 „	12 „

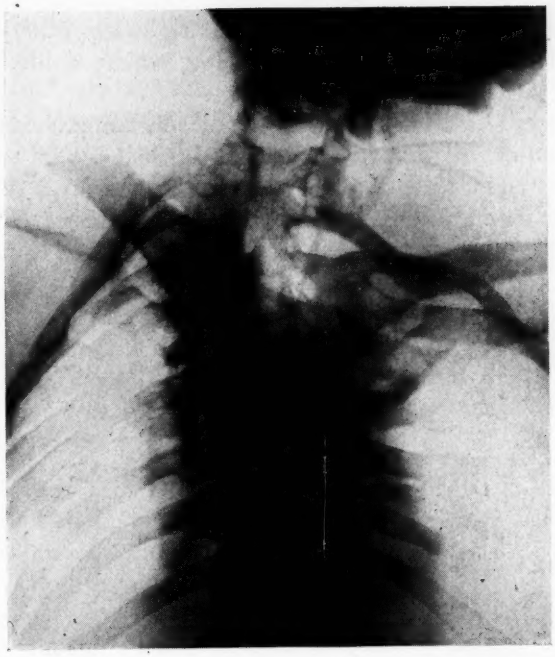
The left foot was a little longer, but somewhat narrower, than the right.

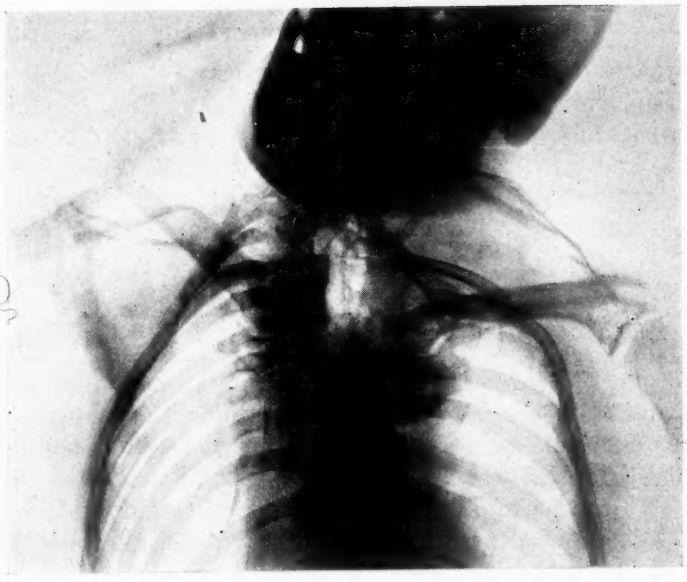
CENTRAL NERVOUS SYSTEM.—No disturbance of mentality was noted. Speech was normal. There was a slight but obvious facial asymmetry, the left half being a little smaller than the right. The palpebral fissures were unequal, and there was a condition of heterochromia iridum present, the left iris being a deeper blue than the right.

Motor System.—Patient was right-handed. The left arm and hand were smaller in all dimensions, but there was no localized muscular wasting. The patient could not abduct his left arm beyond 45°, and internal and external rotation movements were limited. All other movements were possible, but were somewhat weaker on the left side than on the right. There was no disorder of tonus or co-ordination. No involuntary movements were present.

Trunk.—The recti abdominis contracted well, and there was no deviation of the umbilicus on attempting to sit up.

Legs.—The left leg was a little longer than the right, and was held in an attitude of extension and internal rotation. There was some inversion of the left foot. The muscles of the left leg were flabbier and less bulky than those of the right. All movements were possible in both legs, but the left





FIGS. 151 and 152.—Two skiagrams showing the cervico-dorsal torticollis and the characteristic Sprengel's deformity of the left scapula. The irregularity in the outline of the ribs is just visible. The skiagrams are reversed.

leg was relatively weaker than the right. Co-ordination was normal. There were no involuntary movements.

There was no disturbance of tone in the right leg, but there was a definite rigidity of the clasp-knife type in the left leg, which tended to wear off after repeated manipulations.

Sensory System (*Fig.* 153).—Cotton-wool was appreciated normally everywhere; appreciation of pinprick was diminished over the whole of the right leg and the right side of the trunk up to the level of the 8th thoracic segment. Above this level, and up to the level of the 4th thoracic segment, pinprick was felt more plainly, but still not as well as on the left side

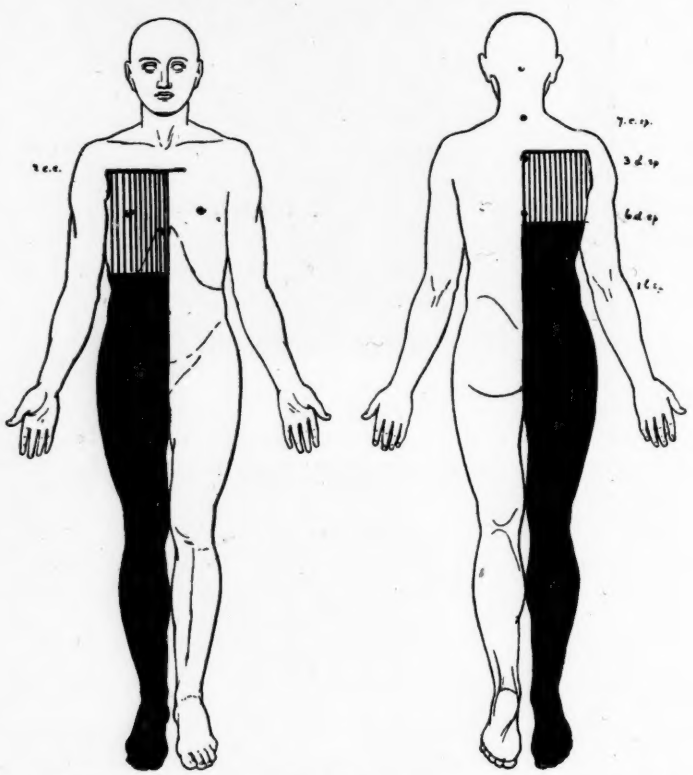


FIG. 153.—Sensory chart indicating area of diminution to heat, cold, and pinprick. There is no loss to light touch.

of the body. Above the level of the 4th thoracic segment, sensation was normal. Hot and cold objects were appreciated less acutely over the same areas in which pinprick was diminished. Postural sensibility was defective in the toes and ankles of both feet. A vibrating tuning-fork was felt as such at the left external malleolus, but not on the right side. There was astereognosis in both hands.

Reflexes.—All the tendon-jerks were present, but were exaggerated on the left side. The abdominal reflexes were absent, and plantar stimulation evoked an extensor response on both sides. Ankle-clonus was obtainable on the left side. There was no disorder of sphincter control.

The Gait.—This was slow and spastic; the left knee was kept extended, the left side of the pelvis was raised, and the left leg circumducted forwards with each step.

No abnormality was detected in the alimentary, respiratory, or cardio-vascular systems. Uranalysis was normal.

Examination of the cerebrospinal fluid (Dr. J. G. Greenfield) was as follows: Clear, colourless fluid; cells, 1 per c.mm.; total protein, 0·015 per cent; Nonne-Apelt test, negative; Pandy's test, negative; Lange, 000000000; Wassermann, negative.

COMMENT.

The case is that of a man of 45, who exhibits a Sprengel's deformity of the left shoulder, together with marked cervical scoliosis and wry-neck. For the past fifteen years he has been subject to weakness, numbness, and pains in the legs, without any disturbance of the sphincters. Examination reveals a condition of paraplegia, the weakness and spasticity being confined to the left side, whilst a hypalgesia to painful and thermal stimuli is obtained on the right side as high as the 4th thoracic segment. Examination of the cerebrospinal fluid precludes any state of spinal-cord compression.

Speculation as to the causation of the paraplegic manifestations in this case raises questions of the utmost interest. Two main possibilities at once present themselves: on the one hand, the paraplegia may be the mechanical result of distortion and traction of the spinal cord and roots by the angular scoliosis; on the other hand, the neurological manifestations may be the result of a coincident congenital intramedullary anomaly of the nature of syringomyelitic cavitation.

In considering the former possibility, the rarity of scoliotic paraplegia must be emphasized. Potts, in his original communication on spinal curvature, made the statement that paralytic signs appear only when the curvature is antero-posterior in direction. This generalization, however, is not strictly accurate. Although paraplegia is a rare sequel of extreme degrees of scoliosis, there are nevertheless cases on record in which the association is beyond question.

Thus Collier[1] showed before the Neurological Section of the Royal Society of Medicine a girl of 16 who, as the result of poliomyelitis at the age of 3, exhibited a severe scoliosis in the mid-dorsal region. Eighteen months before coming under observation her legs began to drag; ten months later flexor spasms appeared; and six months later still she lost bladder control. The girl was then in a state of severe spastic paraplegia in flexion, with a motor and sensory level as high as the 7th thoracic segment. The spinal fluid revealed a loculation or 'partial Froin's' syndrome. In his introductory remarks Collier stated that he had seen four other cases in which severe scoliosis from poliomyelitis had been followed years later by paraplegia.

Elmslie[2] demonstrated two similar cases before the Orthopædic Section of the Royal Society of Medicine. The first case was that of a boy of 18 who developed a spastic paraplegia in extension eight years after the appearance of a post-poliomyelitic scoliosis. There was a sensory loss up to

the level of the 5th thoracic segment. Lipiodol was held up in the spinal theca at the level of the 4th dorsal spine. Elmslie performed a laminectomy of the spines of the 3rd, 4th, 5th, and 6th vertebræ. The cord showed no abnormality beyond a marked degree of stretching. His second patient—a boy of 12—began to develop paralytic signs five years after the appearance of scoliosis. At the time of the demonstration the boy was in a state of spastic paraplegia in flexion. Lipiodol was held up opposite the body of the 1st dorsal vertebræ.

In the present case the symptoms in their full intensity have dated from the dispute in which the arms were twisted and wrenched behind the back. It is certain, however, that mild premonitory symptoms had been present for some years previously. It is conceivable, therefore, that this paraplegia is the result of a prolonged stretching of the cord, and more particularly of its roots in the distorted bony canal, the trauma being the precipitating etiological factor. Such a hypothesis would account for the motor and sensory defects in the legs and trunk. It is difficult, however, to explain the total left-sided hemiatrophy and the heterochromia iridum on such a hypothesis. The objective sensory changes on closer examination become more and more difficult to reconcile with a purely extramedullary lesion; the dissociation of the sensory loss over the right leg and trunk—though well known for many years as a temporary symptom of extramedullary lesions of the spinal cord—is known to have existed in this present case for at least fifteen years. Such a protracted dissociation favours rather an intramedullary origin. Again, the bilateral astereognosis is scarcely explicable on the hypothesis of a scoliotic paraplegia.

It is perhaps easier to explain the symptomatology of the present case by regarding the central nervous manifestations as syringomyelitic in nature. The paraplegia with bilateral extensor plantar responses, the dissociated hemianæsthesia, the bilateral astereognosis, favour such a diagnosis. The facial asymmetry may be the result of a lifelong torticollis, but a heterochromia iridum is almost certainly the result of a prolonged unilateral affection of the sympathetic, and is a manifestation not infrequently met with in syringomyelia. The marked aggravation of symptoms after an injury is a well-known phenomenon in this disease, and may be the result of a hæmorrhage into the syringomyelitic cavity.

The co-existence of syringomyelia with various skeletal anomalies, apart from the characteristic scoliosis and arthropathies, is well known. Thus, one has seen the disease associated with acromegaly (Macbride,[3] Petrén[4]), cervical ribs (Oppenheim, Marburg), and spina bifida (occulta and aperta). The disease has also been described associated with recurrent dislocation of the shoulder-joint (Schrader) and cheiromegaly (Charcot and Brissaud). As far as one has been able to trace, there is no recorded instance of an association between syringomyelia and Sprengel's deformity.

The case is also of additional interest in exhibiting, in common with most cases of Sprengel's deformity, other bony defects in the cervico-thoracic regions. As in the case recorded by Niederle[5], there appears to be a reduction in the number of cervical vertebræ—the so-called Klippel-Feil syndrome. This would partly account for the apparent absence of a neck on inspection

of the patient. The marked torticollis and scoliosis have already been referred to. In Horwitz's[6] series of 136 cases the former deformity was present in 10 per cent and the latter in 47 per cent of cases.

Deformity of the ribs is common in association with elevation of the scapula, and in this instance the second left rib is seen in the skiagram to be of unusual girth, and to articulate with the side of the head of the 3rd rib, while the 4th, 5th, and 6th ribs on the left side are of usual breadth and conformity (*see Figs.* 151, 152).

Unlike Neuhoff,[7] we have been unable to trace any evidence of an heredo-familial incidence in our patient. There is no sign, moreover, of an elevated scapula on the opposite side, although a bilateral Sprengel's deformity occurred in 11 out of Zesas'[8] series of 99 cases.

I should like to thank Dr. Collier for his kindness in permitting me to record this case.

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THE RADICAL OPERATION FOR URETHRAL STRICTURE.

By R. HAMILTON RUSSELL, Melbourne, Australia.

There is perhaps no human ailment that is the source, in the aggregate, of suffering so intense, prolonged, and hopeless as severe urethral stricture under treatment by dilatation; yet dilatation has seemed from time immemorial the best method of treatment that surgical science has been able to offer, and it has never been superseded, nor apparently even seriously challenged. "Dilatation first, dilatation always," has been accepted as the epigrammatic statement of a firmly-established principle governing the treatment of urethral stricture; while operative enterprise has been almost entirely occupied with procedures designed to facilitate or render possible subsequent treatment by dilatation.

There are various tubular channels in the body that are prone to become strictured, to the ruin of their function; but of these there is only one, the abdominal portion of the alimentary canal, that has seemed to lend itself to successful treatment by excision of the stricture with restoration of the continuity of the channel. The special features that have rendered this possible are, first, the peculiar healing properties of apposed peritoneal surfaces; secondly, the mobility and the size of the intestine. None of these attributes are possessed by the male urethra; hence it is not surprising that attempts to excise the stricture and unite the severed ends of the urethra over a catheter have never proved attractive to the large majority of surgeons, nor have the devices usually been of a nature to inspire confidence.

On the other hand, were it possible to devise some plan of excising urethral strictures that should be as straightforward and as certain as excision of an intestinal stricture, this would at once bring about a 'revolution' in the treatment of urethral stricture, and dilatation would be promptly brought down from its 'bad eminence' and would find only a relatively humble sphere of usefulness, very much in the background.

It is precisely this 'revolution' that has already taken place in the practice of a number of leading Australian surgeons, and it is in part the purpose of this paper to explain how it has come about, and upon what it is based. It means necessarily that some observations must have been made revealing attributes possessed by the male urethra which are as distinctive and favourable to the operation of excision as are those that are peculiar to the intestine; and this is so. Such attributes, however, must be entirely different in the two cases, for in dealing with the urethra we have no helpful peritoneum to aid us, and the deep urethra is anything rather than a mobile structure.

In the British Journal of Surgery (1915, ii, 375) will be found the description of a method that I had found successful in the excision of urethral stricture, based upon a principle that seemed to me of great importance, and,

so far as I was aware, entirely new. One author alone, the writer in Binnie's *Operative Surgery* (1915), seems at once to have grasped its significance, stating that in his opinion "this operation promises to supersede all others," and giving an admirable condensed account of it that would seem to be all-sufficient as a guide to an experienced operator. But for this circumstance I should have reproached myself for having failed to make the essential principle of the operation as clear-cut as it can and should be made. My main object in this paper is to describe an amended and greatly simplified technique of the operation; and incidentally to call attention to the special qualities displayed by the urethra—qualities that are its peculiar possession, and which show it to be more richly endowed than even the intestine with attributes that favour the treatment of stricture by excision.

The Special Attributes possessed by the Male Urethra which favour the Treatment of a Stricture by Excision.—These are two: (1) The capacity for spontaneous restoration after it has been slit up, no matter to what extent. (2) The elasticity or extensibility, which is physiologically controlled, and enables the urethra to lengthen and shorten in response to changing conditions of the generative organs. I think I am safe in concluding that the possession of this attribute is highly favourable to us when we desire to sacrifice a short length of the urethra with a minimum of inconvenience. Neither of these singular attributes is possessed by any other tubular channel in the body; and their co-existence in the urethra implies an obligation to examine into means for making use of them.

The Capacity for Spontaneous Repair.—It is the capacity for spontaneous restoration after the slitting up of the urethra that affords the clue to a means by which a stricture may be excised. The 'how' and the 'why' of it is explained in a paragraph which I will transcribe from my earlier paper (loc. sup. cit.):—

> "The urethra when slit up is converted from a tube lined with mucous membrane into a 'riband' covered with mucous membrane; its restoration is to be left entirely to natural processes, and will depend upon the obvious fact that a strip of mucous membrane flanked on either side by raw tissues, which tend naturally to fall together and cohere, will, when this has taken place, be of necessity converted into a tube."

Clearly it should be an easy matter to excise a short length of the urethra by cutting out a portion of the 'riband' and bringing the cut ends together by catgut sutures. The knots of the sutures will all be inside the urethra, and will not be seen again; while the circular urethral wound, protected by means of perineal drainage from irritation by the urine and from contact with the catheter, is found to heal very readily.

The method of operating I am about to describe will be found to differ from that described in my original paper in the following important particulars: (1) The membranous urethra is not sought for and opened in the first stage, and often not opened at all. (2) The channel through the strictured portion of the urethra is disregarded and not looked for. In this way the most tedious and difficult steps of the operation are escaped; and the deep dissection and disturbance of the deep perineum in the search for the membranous urethra avoided altogether.

THE OPERATION.

The patient is placed in the extreme lithotomy position.

STEP 1. (*Fig.* 154).—The angular incision B A C, the apex A being over the central point of the perineum. The deep fascia of the ischiorectal fossa is seen, and must be opened on either side with a blunt instrument; the left thumb and forefinger are introduced so as to grasp the front portion of the external sphincter. This muscle is now severed from its attachment to the bulbocavernosus muscle at the central point and pushed backwards.

(The new method now diverges from the old, in which the next step was the membranous urethra.)

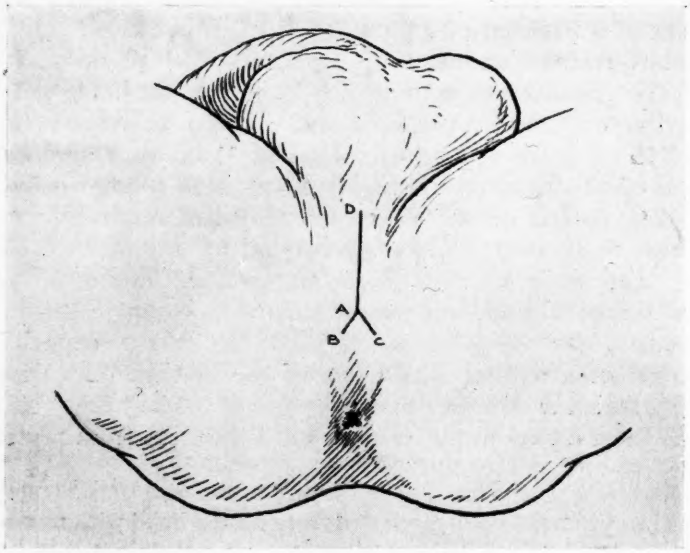

FIG. 154.—The angular incision, B A C, and mesial perineal incision A D. A should be over the central point of the perineum.

STEP 2.—The mesial perineal incision A D (*Fig.* 154), exposing the bulb and several inches of the corpus spongiosum and urethra, but without opening the urethra yet. Define the inflammatory mass comprising the stricture by dissecting it up on either side so as to loosen it in its bed, thus making it easy to lift it *en masse* out of its bed when the moment arrives.

STEP 3 (*Fig.* 155).—Take a full-sized metal bougie, straight for choice, and pass it down to the face of the stricture; clearly the whole of the urethra now occupied by the bougie is in good order and must be preserved. Make a transverse cut into the urethra over the point of the bougie, withdraw the bougie, and continue the transverse cut until the urethra is entirely severed (N.B.: in all its coats) immediately in front of the stricture. The sound urethra will now be entirely detached from the inflammatory mass (*Fig.* 156). During this step some slitting up of the sound urethra may be done if the surgeon thinks it helpful.

STEP 4.—Dissect the inflammatory mass out of its bed on the triangular ligament, working from before backwards and laterally. As it becomes loosened, turn it over backwards so that the dorsal aspect of it, with the urethra, the urethral foramen in the triangular ligament, and the face of the triangular ligament itself, all come into view (*Fig.* 157). The most distal portion of the mass—and that is where the stricture must be—is now nearest to the surgeon; on the upper aspect of the mass will be seen the urethra emerging from its foramen in the triangular ligament, to lie more or less embedded on the surface of the mass for a little distance, before plunging into it at the stricture site.

STEP 5.—To remove the stricture and the inflammatory mass with the minimum length of the urethra. We know that the stricture must be situated at the distal end of the mass, and that it must lie between the distal end (previously called the 'face of the stricture' when *in situ*) and that point at which we can see the urethra entering the mass on its dorsal surface.

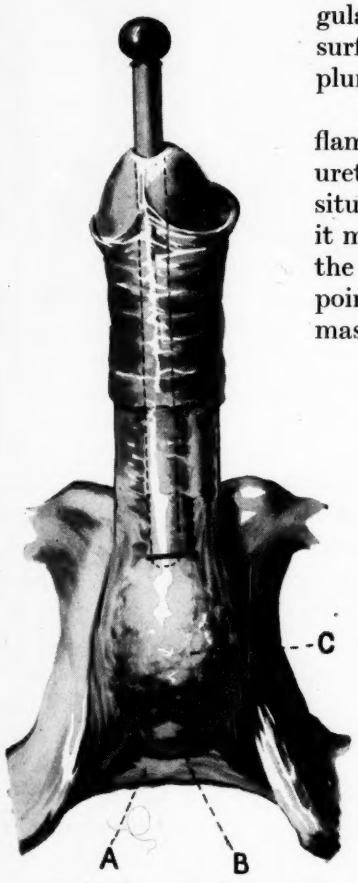

FIG. 155.—A bougie passed down to the face of the stricture, and the first transverse cut made over the point of the instrument. A, Triangular ligament; B, Bulb; C, Stricture mass.

FIG. 156.—The corpus spongiosum and urethra completely divided.

Proceed thus: By a series of sections about $\frac{1}{4}$ in. thick, cut away the distal end of the mass until the open mouth of the urethra (*Fig.* 158) appears, with perhaps a little urine flowing from it; pass a full-sized rubber catheter through it into the bladder, thus proving that the stricture has been entirely removed. We can now clip away the remainder of the inflammatory mass with knife and scissors, and we are left with perfectly sound proximal and distal urethra to bring together across an intervening gap that looks wide, but will prove in reality to be trivial in extent.

STEP 6 (*Figs.* 159, 160, 161).—Preparation for suturing: Both the proximal and (if not already done) the distal portion of the urethra are now freely

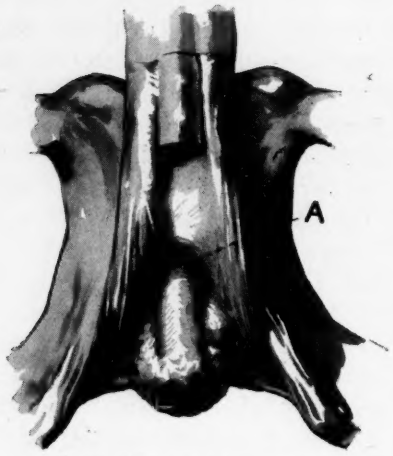

FIG. 157.—The stricture mass raised from its bed and turned backwards, exposing the urethral foramen (A) in the triangular ligament, with the urethra outlined on the dorsal aspect of the mass.

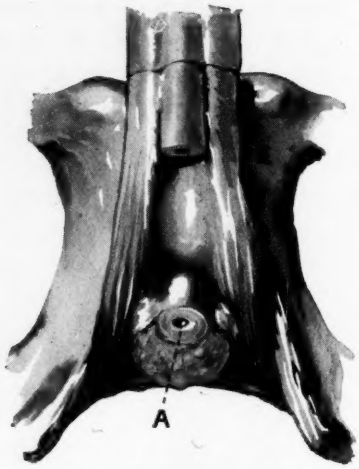

FIG. 158.—The face of the mass, together with the stricture, has been cut away in successive sections until the open mouth of the urethra (A) is exposed.

slit up, so that the interior of the canal is displayed, being now converted into a 'riband' instead of a tube. The ends of the 'riband' are then sutured together with fine chromicized catgut, the knots being on the mucous surface and eventually inside the urethra. In passing the sutures, care must, of course, be taken to include a substantial share of the external coats of the urethra and corpus spongiosum. A good plan I see followed by some of my friends is to put one or two relaxation sutures into the outer coats. A rubber catheter is passed into the bladder and fastened with a suture to the skin; one catgut stitch passes through the skin on either side at the level of the central point of the perineum, and takes up also the anterior end of the triangular flap, thus bringing it into its proper position. (I think this suture may usually be omitted.) A suitable dressing is now applied, and the legs are released and brought down.

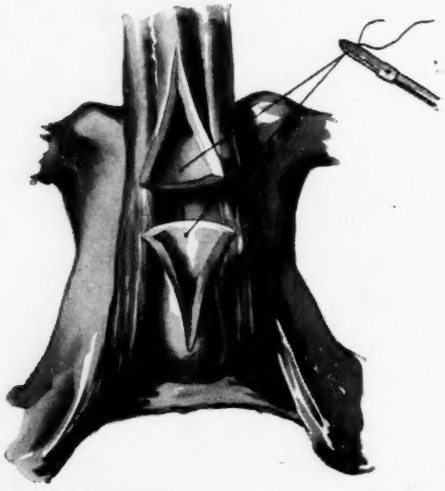

FIG. 159.—The proximal urethra has been cleared of the remains of the inflammatory mass, both proximal and distal portions of the urethra slit up to any extent that may be convenient, and the first suture introduced.

After-treatment.—On returning to bed, a rubber tube is attached to the catheter, and the urine conducted to a bottle suspended from the side of

the bedstead. Each morning, when the dressing is changed, the bladder and catheter are gently washed through with boracic solution. On the fifth day the catheter is removed, and complete healing of the perineal wound will rapidly take place. Frequently the urine will pass almost entirely through the penis during the first twenty-four hours after removal of the catheter.

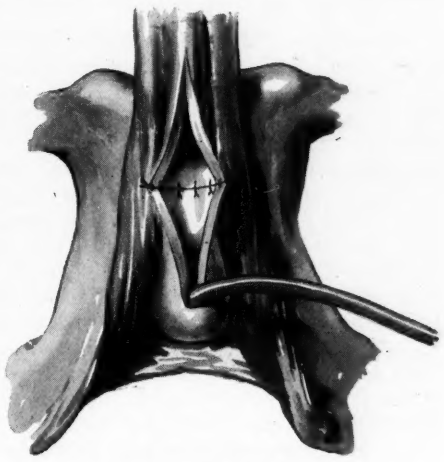

FIG. 160.—The suturing completed and the catheter introduced.

The Passing of a Sound: When should this be done?—It would appear, at the first glance, that the removal of a stricture and the substitution of an annular cicatrix, however accurately brought together by sutures, is likely to prove of doubtful value. The view has, in fact, been urged that in so doing we shall be merely replacing one form of stricture by another as bad. Nothing could be more erroneous than such a contention, and one of the most interesting observations we have made relates to this matter. Early in our experience we used to pass a full-sized instrument at the end of three or four weeks, and to me it was always rather an anxious proceeding; but I do not remember any occasion on which the instrument was arrested at the seat of the former stricture. On the other hand, experience, more or less accidentally acquired, has shown that patients will go for three or four years after this operation without the passage of any instrument, and without experiencing any inconvenience whatever. At the same time I do not think it wise to let a patient alone for so long as this, for the following reason. There is no doubt that the circular cicatrix of the suture line does contract very slowly—so slowly that it takes four or five years to produce any recognizable effect on the size of the stream. Then, however, the patient will probably notice that the stream is diminishing in size, and on instrumental examination it will be found that the opening will only admit a No. 5 or 6 English (I always

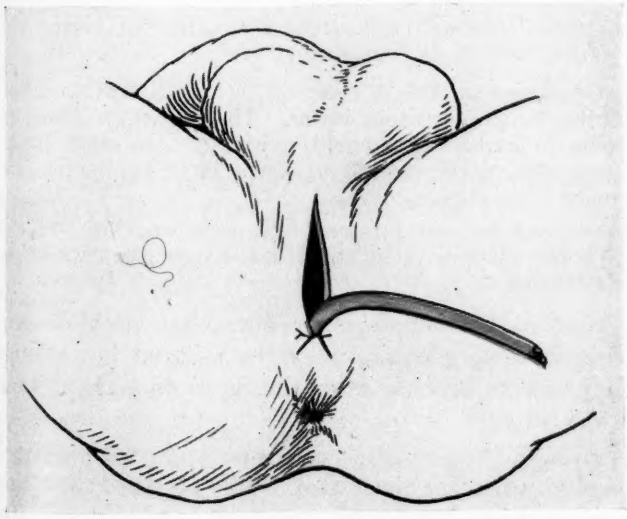

FIG. 161.—The operation finished.

use the Lister graduated bougies). Nothing can be easier than to dilate the stricture up, and he will go on comfortably for another term of years.

In view of these facts, what exactly shall we do ? It is difficult to be precise and categorical, but valuable light is thrown on this important question by a study of the following four cases :—

Case 1.—This case, although not one primarily of stricture but of complete severance of the urethra, is very instructive.

In October, 1910, R. L. B., age 26, sustained severe injury to the perineum by falling across an iron bar. For four weeks after the injury he passed water with great pain, difficulty, and bleeding ; there was, however, no extravasation. On admission to the Alfred Hospital, examination of the perineum revealed a hard defined swelling about an inch in length in the course of the urethra. At the operation it was found that the urethra had been completely divided, and the distal severed end doubled in upon itself into the urethral channel, causing the small but defined swelling that was felt externally, and leaving an inch or more of the urinary channel formed only by the external fibrous sheath of the corpus spongiosum, which sheath had escaped laceration. In order to restore the urethra, the doubled-in portion was freely slit up, and also the end of the proximal torn portion ; the ends were then adjusted and sutured together with catgut. To my great surprise no stricture formed, and in July, 1913, no instrument having been passed for two years, a full-sized bougie passed without any hitch whatever.

I supposed that he was permanently free from further trouble as the result of his injury ; but no. Although I frequently saw him during the next few years, he astonished me one day in 1918, five years after the last instrumentation, by coming to see me with reference to his urinary stream, which seemed to be contracting. On examination, the circular cicatrix admitted a No. 7. Full dilatation was at once carried out, and he has not needed any further treatment, although eight years had passed.

For the purpose of this paper I asked him to visit me on March 25, 1926. There had been no sign of diminution of the stream since the last dilatation eight years ago. I now passed one instrument, Lister 11–14, into the bladder ; there was just sufficient resistance, as the bulbous end passed through the circular cicatrix, to locate its position ; that was all.

Case 2.[1]—R. T., age 38. Operation in Alfred Hospital, Nov. 12, 1917. This man had endured dreadful suffering for three or four years. The operation afforded complete relief, but no persuasion or explanation could induce him to come back to see us. Instead, he was surly and quite thankless. In a later publication[2] I was able to make this further note :—

" 1923. This man has been seen by my former house surgeon, Dr. W. S. Newton. The man states that he is quite well, although no instrument has been passed since the operation " (six years).

I believe that although six years had passed since the operation in this case, this foolish man has only to go on long enough and he will be in serious trouble ; but it will probably prove to be only after the lapse of perhaps ten years from the operation.

Case 3.[3]—W. K., age 55. Operation for stricture in Alfred Hospital, Feb. 12, 1916. No instrument was passed after the operation. He explains that he returned to the hospital on one occasion but failed to see me, and did not go again. He was seen on March 5, 1919, three years after operation. He stated that he had experienced no trouble whatever, but had been mentally apprehensive lest he should suffer through neglecting to have an instrument passed. On examination, Lister bougies 9–12 and 8–11 were arrested at the site of the old stricture ; 7–10 passed through, and dilatation was at once proceeded with up to 12–15. I have not seen him since.

Thus it will be seen that in the case of this man the process of contraction of the circular suture line took three years to reach the size of No. 7, but he had not yet noticed any diminution of the stream.

Case 4.—Medical practitioner, age 54. Operation December, 1921. First instrument passed March, 1926, four and a quarter years after operation. He had noticed some diminution in the size of the stream latterly. Lister 5–8 passed, and dilatation up to 9–12 was at once carried out.

An interesting and useful piece of information seems to emerge from a study of three of these cases in regard to the rate of contraction of the circular suture line. Roughly, it would appear that the rate of contraction will be about two catheter sizes (English) per annum. Estimating the normal calibre of the urethra at Nos. 12 to 14, at the end of the first year contraction will have taken place down to No. 11, at the end of the second year to No. 9, at the end of the third to No. 7, at end of fourth to No. 5; and now for the first time the patient will perhaps notice that the stream is becoming smaller. This is on the assumption that no instrument has been passed during these four years. On the other hand, if the urethra is fully dilated once a year during the first years after operation, the rate of contraction greatly diminishes with age, so that the interval between instrumentations may be extended to several years (cf. *Case* 1, in which after the third year no instrumentation was required until five years later, when No. 7 passed; then after a further interval of eight years there was no diminution of the stream, and No. 11 passed).

Putting the result of these observations into tabular form :—

1. Where no instrument is passed for several years following operation (*Cases* 3 and 4) :—

At end of	1st	year	contraction	to	11	(English)	
„	„ 2nd	„	„	„	9	„	
„	„ 3rd	„	„	„	7	„	Diminution of stream noticed.
„	„ 4th	„	„	„	5	„	

2. Where the stricture is looked after—dilated once per annum during first three years after operation (*Case* 1) :—

During 5 years from 3rd to 8th year, contraction took place to No. 7.
During 8 years from 8th to 16th year, contraction negligible.

Needless to say, there is nothing 'absolute' about these tables. They simply represent exactly what happened in three cases. Further observation is sure to teach us more. Meanwhile I think that, if the above facts be carefully studied, they will be found to furnish a solid basis for very valuable working principles, and I would suggest the following programme as seemingly a sensible one :—

Examine the urethra every twelve months for the first three years, always dilating fully, and be guided by the behaviour of the case as to the future conduct of it. The patient must, of course, have the position explained to him, and be told to come without delay should any diminution of the stream become noticeable; but that is not likely to take place until after the lapse of several years.

The Advantages of Perineal over Per-urethral Drainage.—For one thing, there is always a tendency for a little urine to run down outside the catheter; the perineal catheter conducts such urine outwards through the perineum and away from the suture line in the urethra. On the other hand, penile drainage permits immediate closure of the perineal wound, so that any urine passing into the urethra remains there, with resulting gross contamination of the suture line. This, together with the mechanical disturbance resulting from contact of the catheter with the suture line, is unfavourable to quiet healing. The perineal route of drainage is, in my opinion, indispensable.

Note on Perineal Access to Urethra.—Although this matter is not essentially involved in the operation I have described, it is nevertheless of interest and importance, and should be included in this paper. The urethra from the apex of the prostate to the base of the glans penis may be exposed and laid open through a perineal incision carried forward so as to invade the posterior half of the scrotum. It is done in the following way:—

1. Exposure of the membranous urethra, and the detachment anteriorly of the external sphincter from the bulbocavernosus muscle (*vide supra*).

2. A mesial perineal incision extending forwards to near the base of the scrotum. By retracting the skin forward, the bulb and perineal urethra may be slit up to the extent of 3 or 4 in.

3. If we desire to expose still more of the urethra, it may be done by prolonging the incision forward so as to invade the scrotum to the extent of about 1 in. By again retracting the scrotum forwards we shall be able to expose and slit up the urethra as far as the root of the penis.

4. If the penile urethra is fibrosed and strictured, and we wish to examine the interior of it, this can still be done through the perineal wound by further prolonging the incision until the scrotum is bisected in its posterior half. The finger can then be run along by the side of the urethra, hooked over the dorsum of the penis at its root, and the penis stripped out of its skin and brought naked into the field of operation, the penile skin being now turned inside out like an umbrella. If desired, the penile urethra can now be slit up as far as the base of the glans without wounding the penile skin. There will be no need for suturing any portion of the urethra; the scrotum alone will need a few stitches.

It seems almost unnecessary to add that such an extensive slitting up of the urethra as this would be neither wise nor profitable unless in the presence of very general and severe fibrosis of the urethral walls and corpus spongiosum. Strictures of the penile urethra are quite unsuitable for excision in any case. I have inserted the description of this mode of access because there are cases in which it is valuable, and such cases are not very infrequent.

Note on Urethral Stricture and Syphilis.—It should be borne in mind that practically every case of inflammatory stricture has been dangerously exposed to syphilitic infection, and a blood test should always precede the operation of excision. In one of my early patients a diffuse brawny swelling of the tissues of the perineum followed the operation; secondary

hæmorrhage occurred, and the result of the operation was unsatisfactory and the stricture recurred. I have heard of at least one other case which was attended by the same results; in both cases the blood test for syphilis gave a positive result. While I am not convinced that the phenomena were traceable to syphilis alone, the lesson is nevertheless one that should not be disregarded.

Finally, I desire to acknowledge my special indebtedness to my friends, Victor Hurley and Alan Newton, of the Melbourne Hospital. They have both carried out the operation described in this paper with complete success and satisfaction, while according to me the rôle of privileged collaborator and assistant. I myself have never done the operation as here described, owing to my being no longer in active surgical practice. The semidiagrammatic illustrations have been drawn from my description, with free use of a figure in Spalteholz' *Hand Atlas of Human Anatomy.*

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[2] *Papers and Addresses in Surgery*, 1923.
[3] *Med. Jour. of Australia*, 1919, March 22.

SYPHILITIC ARTHRITIS.

By ALAN H. TODD, London.

(An article based upon a Hunterian Lecture delivered before the Royal College of Surgeons of England on Feb. 6, 1925.)

INTRODUCTION.

Syphilitic arthritis seems to have been recognized as a clinical entity by the ancients, and it was well known and described in the Middle Ages (e.g., Petrus Maetyr in 1485, and Feyrus in 1537); it is mentioned also by a number of the writers at the Renaissance period, and notably by Fallopius, writing in 1574. At a later date this knowledge seems to have been entirely forgotten; John Hunter, writing in 1786, knew nothing about the condition, though he knew that 'mercury cured rheumatism', a fact which is also mentioned by Villelobos in 1498. It was not till 1817 that Sir James Russel recognized syphilis as a cause of arthritis, while Richet wrote the first clear description of the condition, and distinguished it from scrofula, in 1853. Then, in 1886, followed Clutton's classical paper.

Since then, many articles have been written upon the subject, and it is easy to compile a bibliography of three or four hundred references; but though the subject is well known by individual writers, it is not one that is at all widely recognized by the generality of our profession, and the diagnosis is seldom made. Text-books dismiss the subject, as a rule, with a few lines, and certainly the majority of doctors regard syphilitic arthritis as being a rare disease and one that hardly enters into practical diagnosis or treatment.

The particular object of this paper is to show that the disease is by no means so rare as it is commonly thought to be; to direct attention to the fact that it assumes many forms, several of which closely resemble other conditions; to emphasize that syphilis is a cause which should be borne constantly in mind when the diagnosis of a case of arthritis is under consideration, and that in every case the presence or absence of a luetic factor should be investigated and considered; and lastly, it is proposed to describe one or two of the rarer manifestations of syphilitic arthritis, such as are probably not generally recognized as being syphilitic.

Charcot's parasyphilitic arthropathy is well known, and will be dismissed without further mention, while little need be said about Parrot's syphilitic osteochondritis or syphilitic osteitis, both of which are well-known conditions.

Definition.—It is necessary, of course, to draw a very definite distinction between syphilitic arthritis and arthritis occurring in a syphilitic subject. By syphilitic arthritis is meant arthritis due to syphilis. In a good many of

the recorded cases, it is evident that the diseases described were not really due to syphilis, but were simply cases of rheumatic arthritis, gonorrhœal arthritis, or some other condition, occurring in a syphilitic subject. This, no doubt, explains the very variable character of the clinical pictures described and the response to treatment. A true syphilitic arthritis is one which is fundamentally due to syphilis, and is curable by antisyphilitic treament, at any rate if it be diagnosed fairly early. The last is a very important criterion in diagnosis, and is a much more important piece of evidence than any Wassermann test.

Frequency.—Statistics as to the frequency of syphilitic arthritis are absolutely unreliable, for two reasons: (1) Many cases are unrecognized and wrongly diagnosed; (2) Many recorded cases are those of arthritis occurring in syphilis, and not of syphilitic arthritis. Certain facts, however, are suggestive: (1) Parrot says that 5 per cent of all congenitally syphilitic babies have arthritis; Fournier gives the figure as 39 per cent, whilst von Hippel says that 56 per cent of a consecutive series of 77 children with keratitis punctata and other well-recognized stigmata of syphilis had arthritis; in the vast majority of these cases the knees were affected. (2) Schüller states that 70 per cent of all arthritis in children is syphilitic. (3) Paëts says that 63 per cent of natives working upon the Panama Canal suffering from acute arthritis had syphilis, and that the arthritis itself was syphilitic. (4) Lacatère states that 70 per cent of the natives of Morocco have syphilitic arthritis, a fact which is due to the extreme prevalence of syphilis amongst the natives, and their habitual neglect of all forms of treatment.

If it be true that arthritis is as common as has been stated in congenital syphilis, there must be a large number of these cases, persisting into later life, undiagnosed and quite unaccounted for, even after due allowance has been made for the death of a number of these children, and the disappearance of the disease, even without treatment, in a number of other cases. Moreover, this consideration applies only to those cases which are due to congenital syphilis; it takes no account of the comparatively large number of cases due to the acquired disease.

Von Hippel makes this very important observation: *no case of syphilis that has been efficiently treated ever develops syphilitic arthritis.* It seems probable, therefore, that with the highly efficient organization that has now been created for dealing with this disease, in the way of clinics for the treatment of venereal diseases, and the like, this disease will gradually be altogether eradicated; meanwhile, it is essential that every member of our profession (not the specialists only, but the rank and file also) should learn to diagnose the disease in every possible case, in order that dissemination by untreated patients may cease as soon as possible.

The disease occurs in all stages of syphilis, both congenital and acquired. It is a *protean condition;* there can be no such thing as a typical word-picture of syphilitic arthritis. It occurs in many forms, and in very variable degrees of severity, whilst the various forms are not clearly demarcated from one another; transitional forms are found between the various clinical types, so that the whole form a more or less well-graduated series from the mildest cases up to the most severe.

ARTHRITIS IN CONGENITAL SYPHILIS.

In congenital syphilis, arthritis occurs in two forms which are more or less peculiar to this stage of syphilis, and in one form which is common both to the congenital and to the acquired disease. In infants, the typical form is *Parrot's syphilitic osteochondritis*, whilst in older children the well-known '*Clutton joint*' is the common form. In addition to these forms, we meet with certain others which are exactly analogous to the tertiary syphilitic forms met with in adults.

Parrot's Syphilitic Osteochondritis is a juxta-epiphysial inflammation. It occurs in about 5 per cent of congenitally syphilitic children, usually in the first three weeks of life, and practically always during the first three months. It affects the upper more frequently than the lower limbs, which is in contrast with the plastic and other later forms of syphilitic disease. The lesion is a gelatiniform change in the cartilage and bone. These break down to form a greenish-yellow fluid, from which a strongly positive Wassermann reaction is obtainable. The fluid is scanty in quantity. Suppuration may or may not occur, and may or may not be microbic. In many cases complete separation of the epiphysis occurs, and in many instances it is fractured, a fact which is perhaps not as well known as it should be.

The X-ray changes include:—irregularity of the epiphysial line; a widening of the articular space; thickening of the periosteum; decalcification of the region of bone immediately proximal to the cartilage; a 'cupping' of the diaphysis, and a certain irregular density and streakiness of the bone in the region immediately adjacent to the cartilage. The prognosis depends very much more upon the presence or absence of visceral changes than upon the condition present in the bones themselves. Given timely treatment, it is possible to produce a complete resolution in the injured joint; but if the growing-line be much damaged, permanent shortening or deformity may occur; and if the disease occurs in a situation where there is a pair of epiphyses, and one of these be affected more than the other, e.g., at the ankle or at the wrist, considerable deformity may occur later. This condition is somewhat comparable with the marked in-bowing or out-bowing of the elbows which may occur at about 50 years of age or so, a condition sometimes described as Stoll's elbows.

Clutton's Joints.—The joints commonly known by this name were first described by Clutton in 1886. The condition consists in a symmetrical hydrarthrosis affecting both knee-joints in children of 8 to 16 years. The onset is insidious, and there is no pyrexia. A certain characteristic is the absence of pain, which contrasts curiously with the marked swelling of the two knee-joints. In spite of the effusion, the patients are able to walk quite well.

In the vast majority the knees are affected—(41 out of 45 cases). In a good many cases there are eye changes also, the joint disease preceding the eye changes in over 80 per cent. X-ray examination reveals no abnormality beyond the mere distention of the synovial sac, and a certain amount of separation of the bones in consequence. The clinical picture of a case of Clutton's joint is very characteristic, and is, as far as I know, quite

pathognomonic. Wth regard to this disease, it is possible to formulate a clinical aphorism.

CLINICAL APHORISM *No.* 1.—*Bilateral, painless hydrops of the knees in children is due to syphilis.*

The prognosis is favourable; the condition responds very well to treatment, though it is somewhat prone to relapse. With prolonged treatment, however, complete cure usually results, and it is seldom that the disease persists and becomes chronic; at the same time it must be remembered tha in occasional instances this takes place, and it is not possible to tell beforehand which are the cases in which this may occur. Whilst the outlook, therefore, is usually favourable, it is obvious that it is unwise to give too confident a prognosis, especially as a few cases are on record which, beginning as typical Clutton's joint, have ended up with bony ankylosis in spite of the most vigorous and prolonged treatment.

Lastly, there are various types of arthritis which one encounters in congenitally syphilitic children which are exactly analogous to the tertiary syphilis seen in adults. I will postpone consideration of these until I have described the earlier forms which occur in adults; the tertiary forms can then be considered all together.

EARLIER FORMS OF SYPHILITIC ARTHRITIS IN ADULTS.

In *acquired syphilis* arthritis is encountered in various forms and at every stage of the disease. There is no strict delimitation of certain forms to certain stages. Moreover, one form may merge into another, whilst in other cases the forms retain their individuality throughout. One form may appear in more than one clinical stage of syphilis; for example, hydrarthrosis appears in the secondary, the intermediate, and the tertiary stages of the disease. On the whole, however, one may say that syphilitic arthritis is more severe in the later stages of syphilis. It affects the joint structures more deeply, and is very much more destructive, and also more resistant to treatment. Unless it is diagnosed early, and treated vigorously, it is one of the most disabling of joint affections—considerably worse than tuberculosis, for it remains in a half-cured state for many years, and is very resistant to treatment. A tuberculous joint, treated according to the most approved modern methods, usually undergoes complete resolution, or else bony ankylosis; it is only in inadequately treated cases that partial limitation of movement, or partial ankylosis, occurs. A joint which is freely movable is trustworthy, and a joint which is firmly ankylosed is trustworthy. A joint whose range of movement is markedly restricted, or whose range of movement, even within certain limits, is not smooth and painless, is a constant source of disability, unreliability, and perhaps even danger. This is the very condition in which undiagnosed syphilis, or inadequately treated syphilis, is liable to leave a joint.

For example, I have in mind a boy of 12 years, who was diagnosed at first as a case of tuberculous arthritis of the knee-joint, and was treated upon that assumption, though in a rather indifferent manner. The result is that now, seven years after treatment began, he is left with a knee which is neither cured nor yet very active; which refuses to respond to antisyphilitic, or any

other, treatment; which is not ankylosed, and yet which is very far from being freely or painlessly movable; which, in short, is a constant source of annoyance and difficulty to him. It being a case of syphilis, one hesitates to excise such a joint, quite apart from the undesirability of excision at this age for other reasons, such as recurring flexion; the only prospect, therefore, seems to be that this boy will have to wear a caliper until he is grown up, and perhaps even afterwards.

D'Arcy Power's classification of syphilitic arthritis is as follows:—

Secondary syphilis	Arthralgia
	Synovitis — Intermittent
	Synovitis — Chronic
Tertiary syphilis	Gummatous synovitis
	Chondro-arthritis, ulcerating or Virchow's joints
	Tabetic, sclerosing, or Charcot's joints
Congenital syphilis	Suppurative arthritis
	Hydrarthrosis
	Symmetrical serous synovitis or Clutton's joints
	Gummatous synovitis
	Chondro-arthritis, ulcerating, or van Gies' joints

It is not, in my opinion, a complete classification, according to our modern knowledge of the disease, but it is the best that exists, and I quote it in its original form. It must be understood, of course, that it is not to be taken in too arbitrary a sense, and that a certain amount of 'overlap' occurs; that is to say, whilst certain fairly well-defined forms of syphilitic arthritis occur at various clinical stages of syphilis, there are transitional forms also, and the occurrence of the types is not confined to the stages mentioned.

Arthralgia is often regarded as rheumatic. It may appear even before the early rashes; the pain is never severe in character, and would often be more correctly described as an 'ache' than as an actual pain; as in tertiary syphilis, it is very often chiefly nocturnal. It is rather characteristic that gentle passive movement does not increase the discomfort at all; in muscular rheumatism, fibrositis, and similar conditions, movement increases pain, as is well known. The marked increase of discomfort at night, and the absence of increase of discomfort upon movement, will sometimes serve to suggest the correct diagnosis; in some cases, the administration of antisyphilitic remedies causes a temporary increase in the amount of pain, and this usually clinches the diagnosis. A Wassermann reaction confirms this, and the continued administration of suitable remedies produces a rapid and permanent cure. The arsenical derivatives are more effective, as a rule, than the older mercurials. The pathology of the condition is not accurately known, as no one appears ever to have cut into a joint at this stage; but as the range of movements is never restricted, it seems probable that the condition is not one of effusion into the joint-cavity; if a joint becomes acutely distended, there is usually some flexion, and some limitation of movement. It seems more probable, therefore, that syphilitic arthralgia consists in an infiltration of the muscles, fasciæ, etc.

CLINICAL APHORISM *No. 2.—Never diagnose rheumatism in any form until syphilis has been excluded as a possibility in the diagnosis.*

Hydrarthrosis occurs in secondary syphilis in two forms, viz.: (1) *Transient hydrops* (early); and (2) *A later, more persistent form.*

1. The transient early hydrops usually affects the knees, but may be polyarticular. Fluid is abundant, and the synovial membrane is swollen. There are no signs of redness as a rule. Pain is moderate, and gentle passive movement does not hurt, though interosseous pressure may do so. It responds very readily to antisyphilitic treatment, and especially the employment of arsenical drugs. The condition pursues quite a mild clinical course as a rule; but in exceptional instances it is exceedingly acute, and even simulates the presence of suppuration. It is, however, characteristic of the disease, even in this very severe form, that the timely administration of antisyphilitic remedies produces a rapid and complete cure. Pain, tenderness, malaise, and pyrexia—all or any of these may be present or absent.

2. The later and more persistent form of hydrarthrosis may be chronic from the outset, or may become chronic, following an acute onset. This condition may be symmetrical, and then, like Clutton's joint, the condition is extremely suggestive, and practically pathognomonic, of syphilis. It is seldom that the two joints are equally affected at any given moment; as a rule one side starts first, and the other follows later, and under treatment one side gets well sooner than the other. There is a marked tendency to relapse—usually at irregular intervals, though occasionally the relapses are periodic. The disability, as compared with the amount of distention of the joint, is very slight, and so is the muscular wasting. Discomfort is moderate in amount, and sometimes the joints are quite painless. The condition may disappear spontaneously, very much as a Hunterian chancre may, even in the complete absence of treatment. The diagnosis is made upon these characteristics, together with the Wassermann reaction, the presence of other evidences of syphilis, and (most important of all) the effect of antisyphilitic remedies. The presence or absence of a history of syphilis is of very little account; one French writer mentions that of eighteen cases in which the diagnosis of syphilis was well established, not only upon clinical signs, but also by the Wassermann reaction and the effect of subsequent treatment, only one patient admitted infection, even when taxed directly with it. Sometimes, however, the family history may give a certain amount of help.

The Plastic Form of secondary syphilitic arthritis is much rarer than hydrarthrosis. There is, as a rule, a long history; there is thickening of the synovial membrane, and especially of the perisynovial tissues; the whole joint seems swollen, and the swelling has the shape of the joint-cavity; but there is, in fact, very little fluid present, and the greater part of the swelling is due to a general tumefaction of the soft parts about the joint; such fluid as there is, is turbid and thick, but not flaky or stringy. It gives a positive Wassermann reaction in 100 per cent of cases, and the Wassermann reaction in the blood-serum is positive in a majority of cases. It is very difficult to detect any spirochætes in the joint-fluid, but lymphocytes are present in great abundance, whilst polymorphs are in a minority. There is usually a fine crepitation on movement, and a certain amount of pain, though not very much. Pyrexia is absent. The response to treatment is very slow, but most cases seem to recover eventually; they often take one to two years in doing

so. The later the diagnosis is made and treatment is begun, the more chronic will the disease be. The crepitation is no doubt due to the rubbing together of thickened soft parts, for it may disappear altogether after treatment, and does not necessarily indicate that ankylosis is inevitable. The plastic form of syphilitic arthritis is usually monarticular, but may be polyarticular in a patient whose syphilis has been neglected. As a rule a big joint is affected, and usually the knee. The order of incidence of the condition in the various joints appears to be: knees, elbows, mid-tarsal, shoulders, wrists.

No sharp border-line can be drawn between these cases and those in which there is some affection of the bone itself; transitional forms occur, and, if the bone is affected, the disease may terminate in some cases by changes of an osteo-arthritic character.

TERTIARY SYPHILITIC ARTHRITIS.

GUMMATOUS ARTHRITIS.

In the early stages of this condition it is the synovial membrane that is mainly affected, and the joint is distended. At this stage the use of mercury and other remedies will effect a speedy and apparently permanent cure. As a rule, however, actual structural changes, such as fibrosis, etc., are present, and these cases are therefore much less favourable than the preceding forms. Even at this stage, timely treatment is still capable of effecting a cure, but later forms are amongst the most chronic known. Occasionally acute and sudden in onset, they are usually the reverse. The pathological changes may be fibrotic or ulcerative from the very first (i.e., cases of tertiary syphilitic type), or may develop out of secondary syphilitic forms, such as hydrarthrosis or possibly the plastic form. When occurring in children, these joints are sometimes described as van Gies' joints. They simulate tuberculosis very closely, and probably most cases are mistaken for that condition. Reschke mentions a case in which one knee was excised for tuberculosis; an ulcer next appeared in the mouth, and this also was regarded and treated as being tuberculous; eventually, the suggestion of syphilis was made, and a Wassermann reaction proved to be strongly positive; antisyphilitic treatment effected a complete cure, both of the joint and of the mouth, within a few weeks.

The gummatous process may be purely local, or it may affect the whole joint, and any structure of the joint may be affected. The condition may result from congenital or from acquired syphilis, and the pathological picture and process are identical in either case. Axhausen has described two forms, namely, (1) *Synovial;* and (2) *Osseous.*

1. **The Synovial Form.**—Occurring chiefly in children, this disease is usually the result of congenital syphilis. Sometimes there is a history of trauma, which may lead to an erroneous diagnosis of tuberculosis. As a rule the larger joints are affected, the order of incidence being: knee, ankle, elbow, shoulder; but the smaller ones may be affected, e.g., the interphalangeal joints. As a rule the effusion is very considerable in amount, because the patient is able to get about better than he could if the disease were tuberculous, the condition being a painless one. Exceptionally, however,

the pain is great, and in these cases the disease is commonly diagnosed as being tuberculosis. Though called 'synovial', the condition is, strictly speaking, a perisynovitis. The lining endothelium and articular cartilage remain shiny, and it is the outer layers, i.e., the perisynovial tissues, which become thickened. They form what has been described as a pseudo-tumor-albus. In exceptional cases a fungous form of arthritis is present, with local projections of coral-like form into the joint.

It is obvious that the simulation of tuberculosis can be very close, but certain points assist in the differential diagnosis: there is less muscular wasting than in tuberculosis, and, even after years of neglect of treatment, there may be little pain and little restriction of movement, whilst suppuration is practically unknown, and no other complications occur. It is practically inconceivable that a child with a greatly thickened tuberculous joint could go about for any length of time without splintage or other adequate treatment, and yet not suffer grave aggravation of his complaint; and in any case, supposedly tuberculous, in which this condition of affairs is present, the diagnosis of syphilis should be seriously entertained and duly investigated. The presence of an irregular, local, lumpy thickening about the joint should always suggest the possibility of syphilis, though in rare instances tuberculosis gives rise to a similar condition. The diagnosis will rest mainly upon the result of a Wassermann test and the effect of treatment, whilst in some cases a helpful indication will be derived from the presence or later development of other evidences of syphilis, such as periostitis, eye changes, etc. As in all cases of suspected joint syphilis, the joint should be punctured, and the Wassermann reaction tested upon the joint-fluid as well as upon the patient's blood-serum.

Clinical Aphorism *No. 3.—Never diagnose tuberculosis or any other form of arthritis until you have excluded syphilis.*

2. **The Osseous Form.**—When this occurs in young patients, it may be primarily an epiphysitis; the articular surface becomes eroded and irregular, and there may be cavitation in the interior of the bone, together with bony outgrowths and a secondary eruption of the inflammatory process into the joint. In adults, the condition bears a considerable resemblance to an ordinary osteo-arthritis in many cases, and in most cases osteo-arthritic changes eventually appear. X-ray examination reveals the characteristic bony changes, together with distention and some increase in opacity of the soft parts about the joint. Though described for convenience as the osseous form, it must not be assumed from this description that the disease is by any means confined to the osseous system; on the contrary, all the structures which enter into the formation of the joint are usually affected, and the condition is merely described as osseous because it is in the bones that the most marked changes occur. In some instances, the joint changes are merely a part of a widespread disease of the skeleton, and the bones are profoundly affected by tertiary syphilis, and extraordinarily deformed, throughout the whole of their length, or at any rate in parts altogether external to the articulations. It is not uncommon for gummata to break down and discharge externally, in which case obstinate sinuses result.

Sometimes the spine is affected, and there is then a very close simulation of tuberculosis, and as a rule it is only when some more obviously syphilitic lesion makes its appearance (e.g., interstitial keratitis) that the diagnosis of syphilis is even entertained. In the few cases which I have seen the disease has been treated for several years as tuberculous, though perhaps the possibility of its not being tuberculous should have been entertained when it was noted how very little the condition was responding to treatment. Sometimes the simulation of tuberculosis is rendered even closer by the presence of skin lesions which mimic tuberculosis very closely.

Case 1.—M. E. K., age 6, was an in-patient of Guy's Hospital, and underwent twenty-two operations for 'tuberculosis' of the glands of the neck, and was also under treatment for tuberculosis of one hip-joint and the opposite knee-joint. She was seen, in the course of her hospital career, by several surgeons, and no doubt seems ever to have been entertained as to the correctness of the diagnosis. A new house surgeon, anxious to distinguish himself, and with mind unbiased by previous diagnoses, suggested that a Wassermann test should be carried out; this was done, and a strongly positive result was reported. Salvarsan was administered (this was in the early days, when the drug was given intramuscularly), and within ten days the child became completely well.

I had the opportunity of watching her for two years afterwards as an out-patient, and during this time there was no evidence of any relapse. There can be no doubt, therefore, that the disease was syphilitic.

Case 2.—Jimmy W., age 18, had been treated at several of the large hospitals in London for old 'tuberculous' caries of the spine, with the usual hump-back deformity, for which he was wearing a spinal support. The case appeared to be, in all respects, a typical spinal caries, and it was only when evidences of tabes appeared, and he was referred to the Neurological Department, that the diagnosis of syphilis was suggested. Ocular symptoms supervened, and though treatment with '606' was given, only partial relief ensued; but the interesting feature was that the eyes underwent material improvement, and ceased thenceforward to cause any pain at all.

It is not possible to say that this may not have been a case of tuberculosis of the spine associated with syphilitic disease, but it seems more probable, in view of the later history of the case, that the spinal condition also was syphilitic.

In adults, the whole of one large joint is affected—as a rule, the knee. Sometimes there is associated osteitis—this is the commonest form; sometimes the condition is a pure synovitis or arthritis. When bone changes are present, these may be extensive, and affect the whole length of the bone, or at any rate a considerable length exterior to the joint, or the changes may be confined to the intra-articular part of the bone. As a rule the changes are of an osteo-arthritic nature, and consist in the formation of marked osteophytes and other excrescences; occasionally, masses of bone form in the reflexions of synovial membrane, or other soft parts of the joint. In some instances the bone may be locally enlarged, or tender, or both. The disease may erupt into the joint, producing secondary arthritic changes there, or may form external sinuses. Now and again one meets with the development of small cavities in the interior of the ends of the bones, e.g., the external malleolus or the olecranon process. In such cases, there may be considerable pain, before the enclosed cavity bursts to the exterior.

Even in well-established gummatous arthritis, timely treatment is still capable of doing great good, though not so much as in the earlier forms

of the disease. However, it is particularly important to institute thorough and prolonged treatment, because the issue of the case, if this is not done, is so particularly serious. If the treatment be delayed, or inadequate, the usual result is the development either of (*a*) gross osteo-arthritic changes, of the proliferative osteophytic type, or else (*b*) ankylosis. The latter is especially likely to occur if the diagnosis is missed and no antisyphilitic treatment is given. If the articular cartilages be destroyed, bony ankylosis may ensue, and very often gross deformity takes place in the meantime, so that the ultimate result is very severe crippling. Even so, however, this result is to be preferred to the development of a fibrous ankylosis, which is nevertheless a fairly common result of this condition. A fibrous ankylosis is the worst of all possible results of any kind of arthritis, for such a joint lacks the freedom of movement that is necessary to insure immunity from injury, and also the absolute stability which bony ankylosis confers. Moreover, in a case of syphilis, the development of fibrous ankylosis is particularly deplorable, because there is a greater risk in attempting to convert this into a firm bony ankylosis by the performance of arthrodesis than is present in a tuberculous case; there appears to be a definite risk of non-union in these cases, even after a thorough prophylactic course of treatment.

If there is any destruction of articular cartilage, no weight-bearing should be allowed, though movements of the joint need not be excluded. In the case of the lower limb, this means that a weight-bearing caliper should be applied throughout the active stage of the disease, and its use should be continued until the constitutional affection is showing a satisfactory response to treatment, and the joint condition shows a marked subsidence. On the other hand, it is important that the mobility of the joint should be retained, by frequent movements of the joint, when it is not subject to weight-bearing, or else there is an added risk of imperfect ankylosis. In the case of a hip or knee, therefore, the patient should be strictly enjoined to wear his caliper from the moment he rises till the moment he lies down, and to take it off daily, in order to exercise his joint, and put it through its full range of movements.

Guyot mentions a case in which the appearance of this disease directly ensued upon an injury, and in such a case the whole condition might well be regarded as post-traumatic tuberculosis, unless the possibility of syphilis were borne in mind and investigated.

Clinical Aphorism *No.* 4.—*Monarticular arthritis of a deforming type at any age, with or without pyrexia, may be syphilitic.*

A PSEUDO-RHEUMATIC FORM.

This occurs (1) *In Children*, both congenitally syphilitic and those who have acquired the disease; and also (2) *In Adults*.

1. In children it may ensue upon Clutton's joint, either early, or after the lapse of one or two years. Sometimes several joints are involved simultaneously, and, if so, one is worse than another, as a rule. The order of frequency is: knees, ankles, wrists, elbows. In some cases one finds that, as one joint is getting better, another becomes involved; this may lead to a suspicion of rheumatism, properly so-called, but (*a*) the condition does

not respond at all to treatment with salicylates, and (*b*) the rate of evolution of the case is usually much slower than in true acute or subacute rheumatism. A further point in the differential diagnosis is the fact that keratitis occurs in somewhere about 75 per cent of the cases. Probably, however, the most important point of all is the fact that little pain occurs; there may be slight discomfort when the joints are moved, or perhaps the complaint will only be of stiffness. There is distention of the affected joints, but they are not red, and there is little or no muscular wasting, whilst the temperature, often normal, seldom reaches 99°. It appears that the condition is one which is seldom recognized; but the clinical picture of a polyarthritis in a child, without heat, redness, wasting, pyrexia, night-starting, or response to salicylate treatment, and without pain, should make up a pretty characteristic picture. Von Hippel mentions a child who suffered from this disease for five years, and Gilbert quotes one in whom nine joints had been involved in the course of five attacks. If, in a case of polyarthritis in a child, there is no response to salicylates, it is impossible to maintain a diagnosis of rheumatism; on the other hand, if the case should prove to be syphilitic, the response to appropriate antisyphilitic treatment will be rapid and convincing. The following case is of interest in this connection:—

Case 3.—P. J., age 8, limped for about two months, and her mother noticed swelling of the left ankle; she said that the ankle had been hurting her for about two days. A diagnosis of subacute rheumatism was made, and she was given salicylates. She had also a pustular eruption all over her body, resembling acne vulgaris, on and off for about a year. Her family doctor, Dr. Moor, of Westgate-on-Sea, noting that she was an habitual mouth-breather and mentally slow, administered dry thyroid extract, and most of the pustules disappeared in a month or so.

In the following month her left knee developed a swelling, and this was painless; a diagnosis of tuberculosis was made, and the patient was put in a splint. The swelling slowly subsided.

Two months later several pustules just above the left ankle coalesced so as to form three large red patches, as large as a penny or larger, resembling lupus; the skin was thickened and of the characteristic 'apple-jelly' appearance at the edges. Though there was no actual ulceration, it was thought that this might possibly have been the result of the rubbing of the splint. The sore gradually subsided, and was replaced by depressed scars, which have since been slowly replaced by normal skin. Four months later the activity in the left knee seemed to have disappeared, and the splint was discarded. Shortly afterwards a large mass of adenoids was removed. Two months later, the left knee swelled again, and in a further two months the knees became normal again, though some wasting of the left thigh persisted. During the next five months no recurrence took place, and then an effusion appeared in the left knee and in the right ankle.

At this stage the patient came under my notice, and, impressed by the chronicity of the condition, the failure to respond to salicylates, the painlessness of the condition, the remissions of the disease in spite of the absence of immobilization, and the failure of the disease to get worse in spite of the fact that she had been walking about, I strongly suspected syphilis, and dismissed the diagnosis of tuberculosis. A Wassermann reaction proved to be strongly positive, and mercury and potassium iodide were administered. These did no good, and the child was admitted to hospital and given a course of N.A.B. Under this treatment the whole of the swelling rapidly disappeared, and the appearance of the limbs became normal. This was in November, 1924, and no recurrence has taken place since.

2. In adults a number of scattered cases have been recorded in which the simulation of acute rheumatism has been very close. The disease occurs

in young adults; several joints become affected in rapid succession; there is often considerable pain, increased by movement of the joints or pressure upon them; and the joints themselves are very tender, swollen, and red; the temperature may be raised considerably, even up to 103° or 104°. Anæmia and copious sweating complete a picture which is practically indistinguishable, at first sight, from that of acute articular rheumatism. There may be no other signs of syphilis to point to the proper diagnosis. The administration of salicylates, however, does no good, the Wassermann reaction is found to be strongly positive, in some cases iritis or some other manifestation of syphilis may appear, and—most important of all—treatment with neosalvarsan or some other arsenical derivative produces a rapid and permanent cure. Though these cases are rare, it is important to be aware of their existence, and not to persist in the diagnosis of acute articular rheumatism in a case in which the recognized treatment for that condition fails.

Clinical Aphorism *No. 5.—Acute rheumatism which does not react to salicylates is almost certainly syphilis.*

THE PSEUDO-RHEUMATOID FORM.

Occasionally one meets a case of what seems, clinically, to be typical rheumatoid arthritis, with radish-shaped fingers, pyrexia, sweating, pain, debility, intermittent pyrexia with exacerbations and remissions of the disease, etc. The simulation of rheumatoid disease is complete, and it is only by carrying out the Wassermann test in every one of these cases, and treating

Fig. 162.—Pseudo-rheumatoid form of syphilitic arthritis.

those patients who give a positive result with antisyphilitic remedies, that the true diagnosis can be made. In a small proportion of these cases, treatment by '606' or the like effects a rapid and absolutely permanent cure, which seems to prove conclusively that the disease is not rheumatoid arthritis but a form of syphilitic arthritis.

The patient whose hands are illustrated in *Fig.* 162 was seen by me at

Guy's Hospital some fourteen years ago. She had been treated for some time for ordinary rheumatoid arthritis, without the slightest result. At that time, impressed by the case of M. E. K., quoted above, I was making a routine investigation of each and every case of arthritis, no matter what its nature, which I had the opportunity of examining. In this case, to my great surprise, a strongly positive Wassermann reaction was obtained; I regarded the case, quite naturally, as one of rheumatoid arthritis occurring in a patient who chanced to be syphilitic; but, thinking it worth while to follow the matter up, I got her admitted, and treated with '606', which was then a comparatively new drug in this country. The surprise of everyone was very great when the swelling of the hands and all other manifestations of rheumatoid disease disappeared completely within three days; I watched this patient for two years afterwards, during which time the Wassermann reaction remained negative, and no further symptoms of any kind appeared.

Brouardel also mentions the case of a man of 55 years, who contracted syphilis at the age of 17 years, and was married at 23. At 49 years he complained of slight stiffness of the spine; at 53 years he had a pain in the arch of his instep when walking. A year later he was unable to walk, owing to the stiffness of his tarsus. His spine was rigid, especially in the lumbar region; both his ankles were swollen and globular, and X rays showed a bone lesion to be present. Though movements were decreased in range, the joints were not at all tender. His knees now became swollen, and the diagnosis made was chronic rheumatoid arthritis. In view, however, of the history of syphilis, and the fact that one of his children had been born dead, a course of mercury cyanide injections was given, and he began to walk again after six weeks, eventually recovering completely.

The Pseudo-tuberculous Forms.—These have been described in passing, under their various headings, and it is only necessary here to enumerate them. They include: chronic hydrarthrosis; chronic plastic arthritis; chronic gummatous perisynovitis (pseudo-tumor-albus); chronic gummatous arthritis with bone changes and a tendency to ankylosis; chronic quiet ankylosing arthritis; chronic suppurating arthritis.

Suppurative Forms.—In congenital syphilis, aseptic necrosis may occur, and the results of the necrotic process may be discharged as a kind of pus. In other cases, ordinary secondary bacterial infection takes place.

In tertiary syphilis the suppuration is sometimes the end-result of severe gummatous arthritis.

GENERAL DIAGNOSIS.

The Family History must be fully considered; and in particular the history of repeated miscarriages during early married life is significant. In a really typical case it may be found that first of all the mother miscarries, during early pregnancy; later, during the later months of pregnancy; then she gives birth to a child which dies, within the first few days, of obvious congenital syphilis; and later, the children, though puny, survive for a time, only to die of some intercurrent malady. But it must also be remembered that a history of predisposition to tuberculosis may be misleading. For

example, in one case which was eventually proved to be unquestionably one of syphilitic arthritis, the issue was confused for a long time owing to the fact that the patient's mother and sister had both died of phthisis.

Previous Diseases will also be investigated, especially as regards the occurrence of interstitial keratitis, iritis, chronic glandular enlargement, etc. A negative history as regards the primary lesion, in acquired cases, is of no importance whatever, as mentioned above. The absence of any history or evidence of collateral syphilitic lesion, in a case of suspected syphilitic arthritis, need not deter one at all from the diagnosis, for in many cases no other lesions develop, whilst in others they only develop at some later date.

History of Present Condition.—A history of recurring attacks of painless joint-swelling which does not respond to salicylates or other treatment is in itself highly suggestive. If there be pain considerably worse at night (osteocopic), this is still more suggestive. In a person who has had syphilis, the occurrence of a blow may precipitate the onset of an arthritis, just as it does in tuberculosis; and one must guard against the error of over-stressing the importance of this history, and more or less assuming, on this account alone, that the case is one of tuberculosis. In any case in which an arthritis follows a blow upon a joint, in a patient supposed to be syphilitic, the possibility of the arthritis itself being syphilitic should be considered.

The Wassermann Reaction.—

CLINICAL APHORISM *No.* 6.—*The Wassermann reaction should be tested as an absolute routine in every case of arthritis of every degree of severity.*

Neglect of this rule has been the cause of a large number of cases of syphilitic arthritis being overlooked in the past. To carry out a serum reaction in every case of arthritis should be just as much a matter of routine as is the carrying out of an X-ray examination and a test meal in every case of chronic dyspepsia, and no great progress in the diagnosis and treatment of syphilitic arthritis can be anticipated until this rule is universally adopted *and taught.* It is necessary to test both the blood-serum and the joint-fluid, if the latter be obtainable. In a number of cases a positive result will be obtained from the joint-fluid when the blood-serum gives a negative result. This fact also should be more widely appreciated. Even in clinics where the Wassermann reaction is tested as a routine in every case of arthritis, unfortunately one still finds, in many instances, that only the blood is tested. Of course, if the reaction is positive in the blood, this will suffice, but a negative finding in the blood does not negative the possibility of syphilitic arthritis, and a joint-puncture should be made in all such cases. I have repeatedly demonstrated this in cases that were presented to me as being unquestionably 'negative'. In cases of simple syphilitic synovitis, the joint-fluid may be straw-coloured and fairly limpid, though sometimes it is rather thready; it is coagulable, and as a rule at this stage its Wassermann reaction is strongly positive. In the later stages the fluid is usually more curdy, and may be quite thready, or even turbid, whilst under the microscope it is found to contain a fairly large proportion of lymphocytes, i.e., about as many lymphocytes as there are polymorphs. In these cases it is necessary to use an aspirating

needle of quite wide bore, or else no fluid will be obtained for examination. The absence of micro-organisms, both on direct examination and on culture, is, of course, of some importance in the differential diagnosis.

In the later stages of congenital syphilis the Wassermann reaction will sometimes be found to be negative, though the arthritis has not fully subsided; it must not be assumed, however, that the arthritis is not syphilitic, for (1) it may be found at a later date that the Wassermann action is again positive, and (2) antisyphilitic treatment may effect obvious improvement in the condition of the joint. In other words, a negative Wassermann reaction cannot be assumed to exclude the possibility of syphilitic arthritis, in cases of late congenital syphilis, at any rate until the effect of a course of antisyphilitic treatment has been tried. As a corollary to this, one might add that in all cases in which the diagnosis of a case of arthritis has remained obscure, in spite of every possible investigation, it is worth while to try the effect of antisyphilitic remedies. Occasionally a dramatic cure ensues, as, for example, in that of the woman previously mentioned who appeared to be suffering from very typical rheumatoid arthritis of the hands, knees, and other parts, in whom a cure, apparently permanent, was effected after a short course of '606'.

Clinical Details.—In regard to these, certain features may be stressed, and particularly:—

Painlessness, in spite of profuse hydrops, especially when this is associated with a perfectly free range of movement.

Symmetrical synovitis.

General health unimpaired in many cases in spite of prolonged joint affection.

Failure to respond to certain specific drugs, e.g., salicylates.

Persistence of the condition in spite of treatment that would be successful in other conditions—rheumatism, rheumatoid arthritis, tuberculosis, etc.

Osteocopic pain.

Associated evidence of syphilis.

Failure to produce tuberculosis, after injection into a guinea-pig of some of the aspirated joint-fluid, may be of assistance in the differential diagnosis.

X-ray Examinations reveal, as a rule, no abnormality in cases of simple syphilitic synovitis or hydrops. In the tertiary stage of syphilis, however, there are usually well-marked changes, viz., those characteristic of ulceration of the articular cartilages, and inflammation and erosion of the underlying bones. As a rule, as in ordinary osteo-arthritis, the changes are proliferative at the margins of the articular surfaces, and destructive over the pressure-surfaces. As a rule there are irregular and often more or less pointed osteophytes projecting from the edges of the articular surfaces, and upon the whole these are less well defined in outline than are the osteophytes of ordinary osteo-arthritis. Occasionally one sees deep and well-defined local pittting of the bone in syphilitic osteitis, and when this occurs it is very characteristic. In the lower end of the femur, or in the head of the tibia, for example, one may see little rounded cavities, just under the articular surface, of a size that would about accommodate a good-sized pea. In adults there is very often a considerable amount of periostitis associated with the arthritis

properly so-called, and, to anyone who has learnt to recognize by experience the characteristic appearance of syphilitic periostitis in a radiogram, the diagnosis of the arthritis itself is pretty obvious. It is rather difficult to describe the appearance of syphilitic periostitis, but some idea may be given by saying that it generally looks as if the negative had been striped longitudinally, along the diaphysis only, with a soft black crayon, or perhaps an artist's charcoal; and as a rule it is the whole circumference of the bone that is involved, and not only one aspect, as occurs in traumatic periostitis. Moreover, in the latter condition the deposit of new bone is generally more dense and more localized than in syphilitic periostitis, and the pencilling looks as if it had been done with a fine HB pencil rather than with a soft crayon. In other words, the new bone in traumatic periostitis is much more compact in texture than is that seen in syphilitic periostitis.

In children and adolescents the X-ray appearance is usually one of arthritis only, and in many cases, though the changes are obvious enough, it is difficult or impossible to say whether they are those of half-healed tuberculosis or of syphilis. In the knee, for example, one often meets cases in which the articular cartilage has been ulcerated away, the joint surfaces are fuzzy and irregular, there is a certain amount of general porosity of the bones, but no attempt at ankylosis, and one is left in doubt as to the correct diagnosis. In short, X-ray examination is not very helpful in many of these cases, save that it differentiates at once between syphilis and sarcoma.

RESULTS OF ANTISYPHILITIC TREATMENT.

Speaking in general terms, one may say that the earlier the case is diagnosed, the better is the prognosis. Cases of symmetrical synovitis (Clutton's joints) are easily and permanently cured, as a rule, by a short course of antisyphilitic treatment; occasionally, however, one meets one that is very resistant and tends to relapse. The early tertiary lesions often do very well, if diagnosed early and treated vigorously; and even late cases respond, now and again, in a most surprising way. Roberts, for example, mentions three cases that were successfully cured after having been wrongly diagnosed and wrongly treated for a number of years: in two knee cases of six years' and ten years' duration respectively, nearly normal function was recovered, and some necrotic bone foci regenerated entirely; another patient, who had had hip trouble and constant pain for twenty-five years, described himself as being "entirely comfortable" after only ten days' treatment. These cases, however, are fortunate exceptions, and as a rule it must be confessed that, where the syphilis has reached an advanced stage, the cure of the joint will be slower and less perfect, whilst the prospect of a real and lasting cure becomes correspondingly remote.

Some of the most disappointing cases occur in tertiary syphilis in older children. They drift on, becoming neither well nor markedly worse, for a number of years, and relapse from time to time; they are even more chronic than some of the worst tuberculous cases. Sometimes the disease persists in spite of the most assiduous treatment of every kind, and the joint (very often a knee) remains in an exasperating half-healed condition, neither

resolving nor ankylosing, but persisting in a sort of mid-way condition, periodically becoming swollen, painful, or flexed; consequently, these patients have to be treated in caliper splints for a great number of years, for experience has shown that, apart from its undesirability upon general principles, excision is unsafe in these cases, owing to the risk of failure of bony union. Roberts mentions a series of 51 cases diagnosed as tuberculous by competent surgeons, and treated as such for periods up to fifteen years; 26 of these cases were completely cured when appropriate antisyphilitic remedies were administered, so that the advisability of routine Wassermann examination and of the routine administration of antisyphilitic remedies in all cases of arthritis defying diagnosis is abundantly demonstrated. Still, to be quite frank, the results of antisyphilitic treatment at a fairly late stage of the disease are, upon the whole, disappointing, so that our main hope for the prevention of this most disabling disease lies in the direction of earlier and better diagnosis. There is a most impressive contrast between the rapid and complete cure that can be effected in cases of syphilitic arthritis diagnosed in early childhood, and the years of crippling, pain, and deformity that may be inflicted if the diagnosis is missed.

CONCLUSIONS.

1. Syphilitic arthritis is not nearly as uncommon as it is commonly thought to be.

2. There are many forms of syphilitic arthritis, undoubtedly syphilitic, that are not generally recognized, chiefly because the possibility is never entertained when the diagnosis is being made. Some of them closely mimic other commoner conditions.

3. It is very important to diagnose these cases, and to diagnose them early, because tremendous suffering, danger, crippling, etc., are otherwise involved.

4. In *every* joint-case, of whatever kind, the possibility of syphilis should be considered and investigated.

5. *Most important of all, a joint-puncture, with Wassermann reaction and cytological examination of the fluid, and a Wassermann reaction of the blood, should be done as a routine in every case of arthritis of every kind.*

6. If the case remains undiagnosed after all possible investigation, the effect of a course of antisyphilitic treatment should be tried.

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Note.—The papers by Waterhouse, D'Arcy Power, von Hippel, and O'Reilly give additional long bibliographies which have not been included in the foregoing compilation.

THE SO-CALLED BRANCHIOGENETIC CARCINOMA: ITS OCCUPATIONAL INCIDENCE AND ORIGIN.

By RUPERT VAUGHAN HUDSON, London.

Among cases of malignant disease there occasionally occur patients who present themselves with a tumour of the neck and in whom, on careful examination, no discoverable focus of disease can be found. The tumour has the microscopical characters of a squamous carcinoma. In such cases the diagnosis rests between a primary carcinoma of branchiogenetic origin and a carcinoma secondary to a healed or undiscovered focus somewhere in the body.

Many such cases have been recorded in the past, and much controversy has arisen as to their mode of origin. In modern text-books, with the exception of Ewing's *Pathology*[1] and Choyce's *System of Surgery* (Arthur Edmunds[2], Raymond Johnson[3]), the information upon these cases is scanty. No consensus of opinion exists concerning them, and some authors deny that vestigial remnants can give rise to a malignant tumour. The prevalent opinion, however, with regard to these apparently primary tumours of the neck may be briefly summarized as follows: (1) A solid malignant tumour of the neck showing the structure of a squamous-celled carcinoma is most probably secondary to a healed or undiscovered focus somewhere in the immediate vicinity. (2) A tumour originating in vestigial remnant is rare, but occurs in two main types: (*a*) The branchiogenetic carcinoma; (*b*) The so-called endothelioma; the former manifesting itself as a malignant swelling in which fluctuation can be detected, and having on section the structure of a squamous-celled carcinoma arising in a pre-existing cyst; the latter as a solid malignant swelling showing the structure common to the endothelioma of the parotid and the submaxillary glands. The unusually early onset of secondary glandular infection may mislead the diagnostician. Ewing emphasizes the fact that branchiogenetic carcinoma when it occurs is commonly cystic, but admits the possibility of a solid squamous-celled carcinoma of branchiogenetic origin.

PARTICULARS OF CASES.

At the Middlesex Hospital during the last two years there have been no fewer than 10 cases of carcinomatous cervical tumour of obscure origin. Of these cases 4 are still under observation, and may be presumed to be of the same nature; but as the remaining 6 appear to fall into one anatomical, pathological, and etiological group, they are here recorded in detail. The most striking fact which emerges is the occupational incidence of these growths, for they all occurred in miners or grooms.

These tumours each began as a small painless swelling just below and behind the angle of the mandible. The gradual increase in size of the swelling

eventually compelled the patient to seek advice for pain, which was not local, but referred to the temporal region and back of the neck. On examination, the only positive clinical sign was the presence of an ovoid tumour, regular in outline, and firmly fixed to the skin and deep structures. The centre of the growth lay just below and behind the angle of the jaw, and the tumour extended upwards, filling the natural hollow between the upper third of the sternomastoid and the ascending ramus of the mandible, but not interfering with the movement of this structure. In spite of rhinolaryngoscopy, no primary focus was then to be found. Microscopy revealed the fact that the tumours were squamous in origin.

They were all treated by the insertion of radium, contained in platinum tubes 2 to 3 cm. long, and of a wall thickness which varied from 0·3 to 0·5 mm. The tubes carried quantities varying from 10 to 50 mgrm. of radium bromide, and were sometimes used with additional $\frac{1}{2}$-mm. screens.

The following are the records of these cases. The photographs and diagrams (tracings of actual photographs) are so arranged that the treatment and clinical course of each patient may be more easily and rapidly followed.

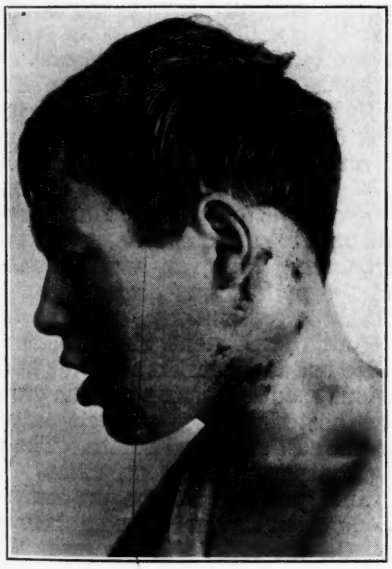

FIG. 163.—*Case* 1. July 21, 1924. Left side, showing the tumour site measuring 9 × 6 cm. The stitches mark the insertion of the radium tubes.

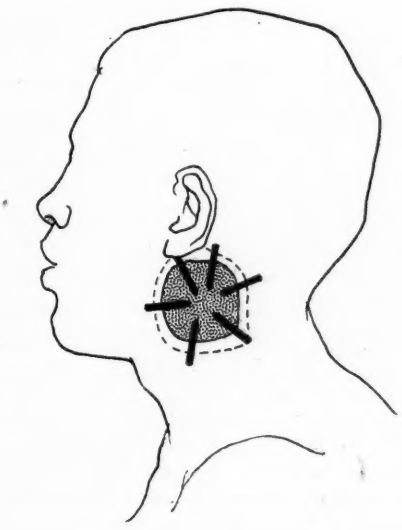

FIG. 164.—*Case* 1. Sept. 1, 1924. The tumour is smaller. Three tubes of 25 mgrm. and three tubes of 50 mgrm., without additional screens, were inserted radially.

Case 1. (Under the care of Mr. A. E. Webb-Johnson.)—E. T., a boy, age 19, who had been occupied as a miner for the last five years. In July, 1924, a year before admission, he had noticed a painless small swelling behind the angle of the jaw on the left side. On admission he presented a large fixed, non-ulcerated mass in the left post-ramal recess. This mass was smooth and regular, and measured 9 × 6 cm. (*Fig.* 163). Below it, and at the crossing of the posterior belly of the omohyoid beneath the sternomastoid, was a small, soft, mobile gland. The only indication of a primary focus was a suspicious roughening in the posterior nares.

The microscopical picture showed the growth to consist of large masses of carcinomatous cells of squamous origin invading lymphatic tissue. No keratinization and no prickle cells were present.

TREATMENT.—Three tubes of 50 mgrm., plus three tubes of 25 mgrm., were placed ¼ to ½ in. below the skin surface, and the tubes were distributed radially at the periphery of the tumour for twenty-four hours. No screens were used. The sentinel gland was excised.

Seven weeks later the irradiated area was flat and healed, but there was a suspicion of growth remaining at its upper pole and deep in the carotid triangle. In the corresponding site on the right side was a soft mass of fixed glands at the superior cornu of the hyoid bone. The original site was re-irradiated for thirty hours with six unscreened tubes, each containing 50 mgrm. (*Fig.* 164). These were placed ¼ to ½ in. below the skin surface, and radially distributed at the tumour periphery between the scars of sites of the application of the former tubes. The soft mass of glands on the right side was surgically removed, and 100 mgrm. of radium were laid in the wound for thirty hours.

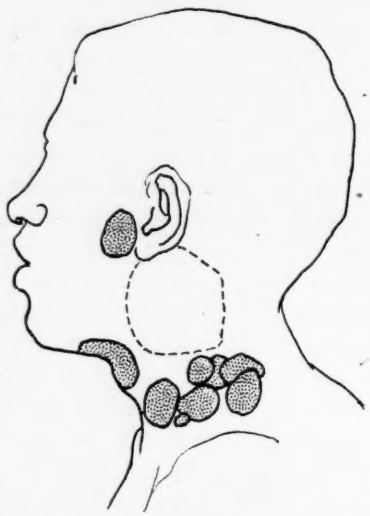

FIG. 165.—*Case* 1. March 1, 1925. Left side. The original tumour site free of growth. The glands below it greatly increased in size and number. The pre-auricular gland further enlarged. Growth is filling the posterior nares and left antrum.

AFTER-HISTORY.—Two months later, and four months from the time when first seen, the irradiated site on the left side was a firm, flat, fibrous but flexible area, with no trace of growth. Above it, the pre-auricular gland was enlarged to the size of a hazel nut, soft and just movable, while below it, and at the site of excision of the sentinel gland, were three large, soft, mobile glands. On the right side in the post-ramal recess, the site of radio-surgical interference, there was a large fixed mass measuring 6 × 5 cm. The mouth was able to be opened only 3 cm. Mouth-breathing was definitely established, but, owing to the difficulty of access, a rhinological examination and report were deferred until an anæsthetic was given. No further treatment, however, was considered advisable, and the patient was discharged.

Five months later, and nine months from the day of the initial treatment, the patient was re-admitted. He had lost considerable weight and there was obvious mouth-breathing. The growth was filling the posterior nares and left antrum, and bulging in the soft and hard palate. The left side of the face was firmly œdematous, obscuring the parotid from palpation. The irradiated site on the left side was still free from growth, but the pre-auricular gland above it was greatly enlarged, while the left subclavian triangle below it was full of large, soft, mobile glands (*Fig.* 165). On the right side the mass in the right post-ramal region now measured 9 × 5 cm., and below it were several large, soft, mobile glands. Swallowing was difficult and breathing stertorous, both no doubt due to the post-nasal growth.

Death occurred at home from inanition two years after the onset of the disease and eleven months after the initial treatment. No post-mortem was available.

The primary lesion here was apparently in the posterior nares, but did not make itself clinically manifest until fourteen months after commencement of the enlargement of glands draining the area.

The clinical course of this case differs from the others of this series. The primary focus was paramedian, and, owing to the local extension of the growth towards the middle line and the lymphatic decussation of the site,

the cervical glands were invaded on both sides of the neck. With the exception of this fixed original mass, the cervical glandular enlargements were always discrete, soft, mobile, and large, but were non-adherent (except slightly to one another) to surrounding structures. Clinically they mimicked the glandular enlargement of Hodgkin's disease.

As regards treatment, several valuable lessons were derived from this case. It was possible, without skin damage or necrosis of subcutaneous tissue, to apply three tubes of 25 mgrm. and three tubes of 50 mgrm., unscreened, for twenty-four hours radially at the periphery of a circle 5 cm. in diameter. Seven weeks later it was possible, again without damage to the skin or subcutaneous tissue, to apply six tubes of 50 mgrm., unscreened, for as long as thirty hours radially at the periphery of the same circle. In both applications the tubes were $\frac{1}{4}$ to $\frac{1}{2}$ in. below the skin surface. After the first dose growth was active in the depth of the tumour and at its upper pole, both areas corresponding to the site of least irradiation. The subsequent dose was introduced during the stage of retardation, and, as a consequence, the growth further retrogressed and eventually disappeared entirely. In comparison with previous dosage used this is enormous, but was only just sufficient to effect its purpose, and did so without damage to normal structures.

On the right side, *where radium and surgery were used in combination*, the results were not to be compared with the result obtained by irradiation alone upon the left side, a fact which confirms the writer's impression that trauma of a malignant site before irradiation is harmful, whether the trauma is surgical, infective, or caused by the unintentional massage by repeated examination. Procedures likely to destroy the normal healthy tissue at the periphery of the tumour will greatly reduce the value of subsequent immediate radium therapy.

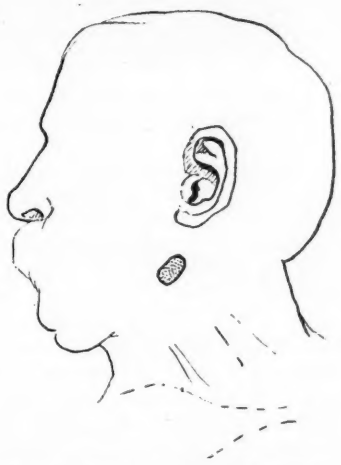

FIG. 166.—*Case* 2. April, 1924: onset.

Although the original mass, 9 × 6 cm., was apparently destroyed *in vivo* by radium, there was no immunizing effect from the dying cells of the region.

Case 2. (Under the care of Mr. Sampson Handley.)—M. S., age 65, had been occupied as a miner all his life. In April, 1924, eight months before admission, he had noticed a painless swelling below and behind the angle of the jaw on the left side (*Fig.* 166). He presented himself with a large, oval, non-ulcerated, fixed mass, measuring 12 × 11 cm. (*Fig.* 167), whose centre lay in the left post-ramal recess, but extended to the mastoid process above, to the level of the thyroid below, and laterally into the posterior triangle. In the centre of the tumour the skin was red and shiny; fluctuation could be detected. The patient's general condition was fair, but he suffered great pain in the head and back of the neck, preventing sleep. He had lost 2 stone in the last six months. No other glands were clinically involved, nor was any primary focus to be found.

TREATMENT.—Under an anæsthetic a quantity of broken-down growth and brown semi-fluid material was removed. Four tubes containing 50 mgrm. of radium

bromide and screened by additional platinum 0·5 mm. thick were placed radially in the centre of the growth for twenty-four hours, and a portion of the growth was removed for microscopy. (*Fig.* 168.)

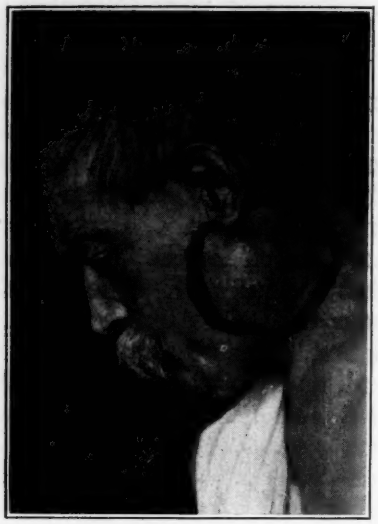

FIG. 167.—*Case* 2. October, 1924. Site of tumour measuring 12 × 11 cm.

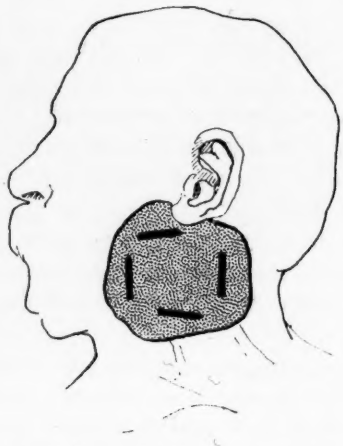

FIG. 168.—*Case* 2. October, 1924. Treatment: a quantity of broken-down growth evacuated, and four 50-mgrm. tubes of radium inserted.

The microscopical section showed a squamous-celled carcinoma arranged for the most part in fine intercommunicating strands. Definite prickle cells could be found, but there was no keratinization. Numerous mitotic figures were present.

AFTER-HISTORY.—The patient was discharged; he was still in pain and only slightly relieved by treatment. A letter was received to state that he had died in April, 1924, a year after the onset of his symptoms. No post-mortem was available, and a further search for a primary focus was therefore not possible.

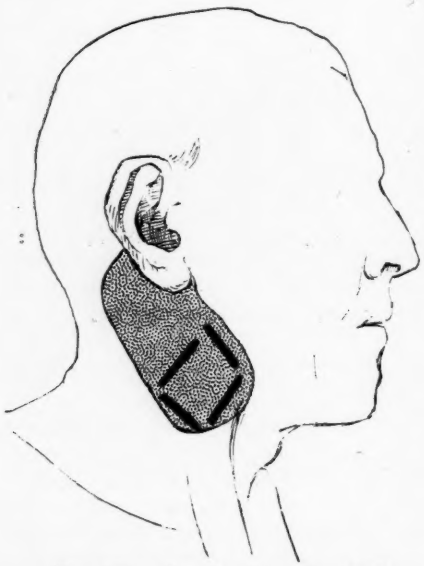

FIG. 169.—*Case* 3. December, 1924. Four 50-mgrm. radium tubes arranged round the caudal half of the tumour.

In this case radium was used with the hope that the pain might be relieved; unfortunately the alleviation of symptoms was not very marked. The case is illustrative of the later stages of these growths and their tendency to remain localized at the site of origin.

Case 3. (Under the care of Mr. Eric Pearce Gould.)—C. R., age 55, had worked with horses all his life. In July, 1924, six months before admission, he had noticed a painless swelling behind the angle of the jaw on the right side. He presented himself with an obvious cervical swelling, oval, regular, and firm, measuring 6 × 9 cm., and situated in the right post-ramal recess. It was adherent to the skin, which showed

peau d'orange over the centre of the growth; the skin was intact. It was adherent to and fixed to the structures beneath, and in the sternomastoid muscle at that level. It was not adherent to the jaw, nor did it interfere with that structure's mobility. No glands were palpable elsewhere, and no primary focus was found either locally or elsewhere in the body. On rhinolaryngoscopy, no primary focus was to be seen.

FIG. 170.—*Case* 3. July, 1925. Showing increase in size of tumour. Note irradiated site apparently still free of growth.

TREATMENT.—Four tubes of 50 mgrm. of radium bromide, unscreened by additional platinum, were buried in the form of an open square round the caudal half of the tumour for sixteen hours (*Fig.* 169). The upper half was not irradiated. A portion of the tumour removed proved microscopically to be a squamous-celled carcinoma. Prickle cells, but no cell nests, were present. In three months the irradiated half of the tumour had shrunk considerably; the upper half had slightly increased in size. Further treatment was refused by the patient owing to fear of losing his present work.

AFTER-HISTORY.—In a further three months the region irradiated was still smaller than when first seen, but the non-irradiated area had quadrupled its size (*Fig.* 170), and the whole mass was now 18 × 27 cm., not ulcerated, but adherent to all the neighbouring structures, producing a facial palsy and interfering with mandibular movement. The condition was too far advanced for any further treatment, and the patient was admitted to the inoperable cancer wards, where he died in November, 1925, fourteen months after the first onset of symptoms.

FIG. 171.—*Case* 3. November, 1925. The site of primary growth. An ulcer and papilloma in the right pyriform fossa. (The œsophagus has been slit on its dorsal aspect.)

At the post-mortem, externally the growth was found to have extended peripherally, while in the centre the skin was invaded and showed an ulcer 3 × 3 cm. Extension had chiefly taken place along the carotid vessels to the base of the skull, and solid growth was bulging the right tonsil and right nasopharynx towards the middle line, but the mucous membrane was not ulcerated. Growth towards the clavicle had not progressed beyond the thyroid level. The primary focus was found, and proved to be in the right pyriform fossa (*Fig.* 171), where there was a small shallow ulcer, 2 × 2 mm., having just above it a small, soft, sessile, malignant, papillomatous growth measuring 5 × 5 mm. No other metastases were found. Death was due to bronchopneumonia. Microscopically both the ulcer and the papilloma proved to be definite squamous-celled carcinoma.

Only half the actual mass of this tumour was encircled by radium. The tubes were concentrated into a comparatively small area and for a short time. In

three months the area of tumour actually irradiated had shrunk considerably. This tumour therefore was susceptible to radium. The upper half of the tumour, which was juxta-tubal but not encircled, was growing, but, judging by the rate of growth from onset to admission (five months), at a much reduced rate. We have therefore a decrease in the encircled area and retardation of the growth at the periphery. In a further three months (six months since the day of treatment) we have, in place of the original tumour, a mass roughly three and a half times the original size, lying in the form of a crescent and apparently originating in the upper half of the tumour; growth has therefore become suddenly rapid.

Case 4. (Under the care of Mr. W. Turner Warwick.)—R. L., age 62, had been occupied as a groom all his life. In February, 1924, he noticed a painless swelling behind the angle of the jaw on the right side. He presented himself with an obvious cervical tumour, a firm, regular mass 9 × 6 cm., situated in the post-ramal recess on

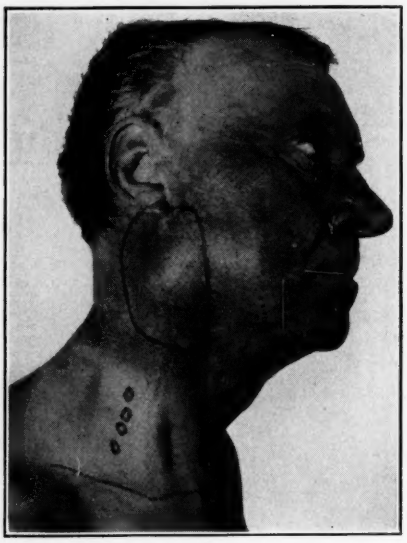

FIG. 172.—*Case* 4. December, 1924. Site of tumour and glands. The dotted lines mark the horizontal ramus of the mandible, the anterior border of the masseter, and the clavicle.

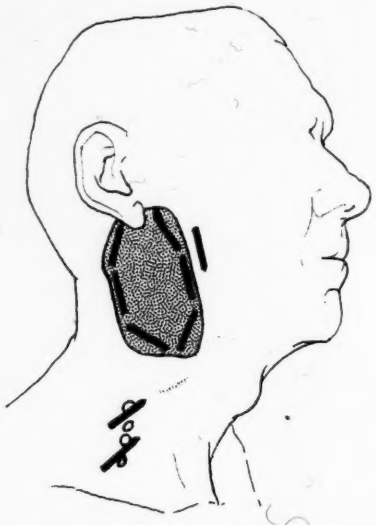

FIG. 173.—*Case* 4. December, 1924. Six tubes of 50 mgrm. and one of 90 mgrm. arranged parallel to and within the tumour periphery. Glands treated by two tubes of 37 mgrm.

the right side. It was adherent to skin over its centre, *peau d'orange* being present. The skin was intact and non-ulcerated. It was adherent to the carotid sheath and fixed in the sternomastoid muscle. It was not adherent to the ramus of the jaw, nor was that structure interfered with as regards its mobility. There were four small glands parallel to and just behind the posterior border of the sternomastoid, the centre gland being just above the posterior belly of the omohyoid muscle (*Fig.* 172). These glands were discrete, firm, mobile, and easily palpable. There was one soft gland in the right axilla. No primary focus could be found on rhino-laryngoscopy.

TREATMENT.—Six tubes containing 50 mgrm. of radium bromide and one tube of 90 mgrm., without additional screens, were inserted for thirty hours (*Fig.* 173). The tubes were buried ¾ to ½ in. beneath the skin, and arranged round the growth, but ¼ in. inside its palpable margin. Two tubes each containing 37 mgrm. of radium

bromide, without additional screens, were placed for eighteen hours over the mobile, discrete glands in the neck. The time was shortened in this case to eighteen hours, as it was impossible to implant the tubes at a deeper level below the skin than $\frac{3}{16}$ in. Convalescence was good and uneventful. During the patient's five days' stay in hospital there was a fair reaction and slight increase in the size of the tumour, as is often the case at first.

The section showed a squamous-celled carcinoma involving lymphoid tissue. The cells were arranged in strands; definite prickle cells were present, together with keratinization and cell nests. Mitotic figures were numerous.

Six months after treatment the patient attended hospital and it was found that there was marked improvement. The skin was epilated, smooth scar tissue. The sternomastoid was movable in its whole length; all that could be felt was a hard, fibrous mass at the junction of the upper third and middle third of the sternomastoid on its medial edge. The size of this fibrous mass was about 2 × 2 cm. and its position was opposite the angle of the jaw. The glands had disappeared, but there was slight induration present. There were no other glands palpable elsewhere, and no signs of mediastinal involvement.

The patient was advised to seek admission for second rhinolaryngoscopical examination, and further radium treatment. It is interesting to note that the residual growth of 2 × 2 cm. was situated at the site of minimum radiation. On re-admission four tubes containing 50 mgrm. of radium bromide, without additional screening, were placed as follows: 50 mgrm. parallel to the posterior edge of the sternomastoid; three tubes of 50 mgrm. placed parallel to one another and horizontal to the sternomastoid muscle. The intervals between the tubes were 1·5 cm., and their depth below the skin surface was $\frac{1}{4}$ in. The duration of exposure was twenty-four hours.

For the following subsequent history of the case I am indebted to Dr. Goone Tilleke, the patient's doctor and the Medical Superintendent of the Highgate Hospital.

A week after leaving hospital, and eight days after the second application of radium, the patient developed very severe pains in the small of the back; these pains were excruciating, and radiated from the sacrum to the lower limbs. He was admitted to the Highgate Hospital, where he developed gangrene of both feet. A local recurrence developed at the irradiated site. Death occurred in May, 1925, ten months after the onset of disease. No post-mortem was performed, but death was attributed to his general condition, and not to the local recurrence.

Progress in this case had been satisfactory up to the time of the second irradiation, and the patient's sudden death after treatment was unexpected. It is evident that the first dose of irradiation was not sufficient to arrest the disease; whether the second irradiation was the accelerating cause or a contributory factor to this comparatively sudden death, it is difficult to state.

The re-irradiation of a region which has been treated by radium some months previously is always a difficult problem. The normal tissues and skin are less tolerant, but the neoplastic cells more tolerant, of irradiation; further, these cells are for the greater part lying in a mass of fibrous tissue which no doubt acts as an additional screening agent. It seems extremely important to deal with a given focus in one single dose, and, if repetition is considered likely, to repeat the second dose within a few weeks of the first, and preferably before the patient leaves hospital.

Case 5. (Under the care of Mr. Sampson Handley.)—G. K., age 58, had been occupied throughout his life as a groom. In November, 1924, five months before admission, he noticed a small painless lump about the size of a large marble behind the angle of the jaw on the left side. Recently he had suffered pain in the temporal

region. He presented himself with a round, hard, elastic, non-ulcerating, firm lump opposite and behind the angle of the jaw on the left side (*Fig.* 174). This tumour lay beneath the sternomastoid in its lateral half, and was intimately adherent to

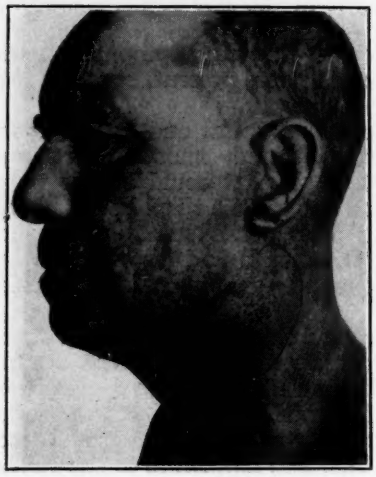

FIG. 174.—*Case* 5. March, 1925. The tumour site is outlined, and the dotted line marks the ramus of the mandible and anterior margin of the masseter muscle.

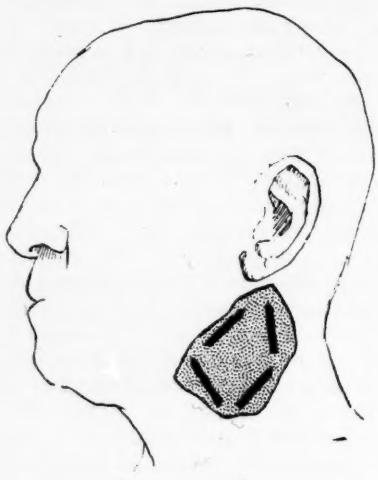

FIG. 175.—*Case* 5. March, 1925. Four tubes of 50 mgrm. inserted parallel to and within the periphery of the tumour.

that muscle. It was adherent to the skin, and was just movable laterally upon the carotid vessels, but not in their length. It measured 6 × 5 cm. No other glands were palpable and no primary focus could be found. A portion was removed for microscopy.

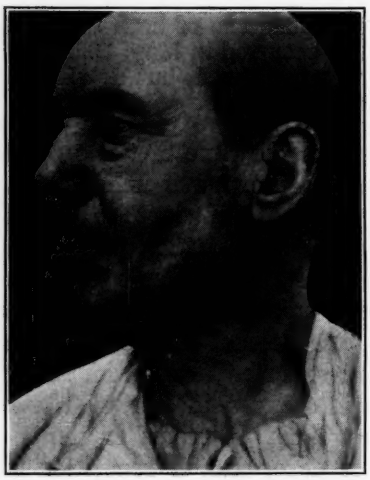

FIG. 176.—*Case* 5. April, 1926. There is no evidence of recurrence.

TREATMENT.—Four tubes each containing 50 mgrm. of radium bromide were placed just within the periphery of and encircling the tumours (*Fig.* 175). The duration of the exposure was twenty-four hours. No additional screens were used. The microscopical section shows a squamous-celled carcinoma invading lymphoid tissue; the malignant cells lay in irregular masses. Definite prickle cells were present, but no cell nests were to be seen.

AFTER-HISTORY. — Up to the present time (twelve months since treatment) all that remains of the tumour is a small fibrous mass, 1 × 1 cm., situated in the sternomastoid muscle and corresponding to the centre of the tumour (*Fig.* 176). No primary focus has yet been found.

This growth was the smallest and earliest of these series, and as a consequence irradiation was more likely to be effective. In the photograph of this patient the tumour appears a little lower in position than actually was the case; but invasion of the sternomastoid had taken place earlier, and was more marked than in the other tumours in these cases.

Case 6. (Under the care of Mr. Sampson Handley.)—T. R., a male of 66, who had been occupied as a groom and occasionally as a gardener all his life. In October, 1924, he noticed a painless swelling just behind the angle of the jaw on the left side. In July, 1925, he found difficulty in swallowing, and was admitted to a hospital, where the tumour was removed.

In October, 1925, three months later, he was admitted to the Middlesex Hospital with an irregular swelling in the post-ramal recess on the same side, extending from the cartilage of the ear above to the superior cornu of the hyoid bone below. The recurrence was hard, irregular, and nodular, adherent to the skin, but appeared just movable on the deep structures. It was not adherent to the ramus of the mandible. The left cord was paralysed and lay in the cadaveric position. There was some hoarseness of voice and difficulty in swallowing. No primary focus could be found on rhinolaryngoscopy.

TREATMENT.—The left sternomastoid, together with the fascia and glands, were removed, but a portion of the tumour was of necessity left behind. Radium was inserted at the time of the operation as follows: Two tubes of 50 mgrm. deep to the ascending ramus of the mandible; one tube of 50 mgrm. anterior to the masseter muscle at the angle of the jaw; one tube of 50 mgrm. at the site of the omohyoid tendon; one tube of 25 mgrm. at the sternoclavicular joint. All the tubes were screened by additional 0·5 platinum. The duration of the exposure was twenty-four hours.

Microscopically the section showed a carcinoma invading lymphoid tissue. The cells appeared to be squamous in type and lay in large masses. No cell nests nor prickle cells were seen. Mitotic figures were present, but not numerous.

AFTER-HISTORY.—The patient only recently left hospital; he is at present still under observation.

This case was the only one of the series in which dysphagia was noticed in the early stages. No doubt its presence signified that spread was occurring, chiefly towards the pharynx, and that there was proliferation of growth in the conduits between the concealed primary focus and the main gland affected. The early recurrence after an apparent complete removal is significant.

COMMENTS.

The Anatomical Site of the Tumour.—In all these cases the cervical tumour arose at the site of the jugulo-digastric gland. As the site of the gland has a great bearing upon the etiological and pathological course of these tumours, it may be useful to note a few of its essential anatomical features.

This gland is large, oval, and flattened, and measures on the average 2·5 cm. in length. It is situated upon the medial aspect of the great jugular vein, having its upper pole at the lower border of the posterior belly of the digastric muscle, and its lower pole opposite the tip of the superior cornu of the hyoid bone. It is placed, therefore, at a site which may be called the 'critical point of the neck', since here—within the space of 2 to 3 cm.—arises the vascular supply to the structures derived from the first five arches, and to this point converge not only the greater part of their venous drainage, but, what is more important, the lymphatic drainage also.

Its importance appears to be due to the fact that it lies in the route of, and forms the main collecting station for, the lymphatics derived from the external auditory meatus, the Eustachian tube, the nasopharynx, the tonsil, the tongue, and the epi-laryngeal structures, the pyriform fossa especially. The main routes of drainage apparently follow the routes of the ascending

A SUMMARY OF SIX CASES OF APPARENT PRIM

Case	Name	Age	Occupation	Duration of Occupation	Date of Onset	Date of Admission	Size of Tumour on Admission	Duration of Disease before Treatment	
1	E. T.	19	Miner	5 years	July, 1923	July, 1924	9 × 6 cm.	12 months	Carcin no ce
2	M. S.	65	Miner	All his life	April, 1924	Oct., 1924	12 × 11 cm.	6 months	Carcin prickl
3	T. R.	55	Groom	All his life	July, 1924	Dec., 1924	9 × 6 cm.	5 months	Carcin prickl
4	R. L.	62	Groom	All his life	July, 1924	Dec., 1924	9 × 6 cm.	5 months	Carcin cells
5	G. R.	65	Groom	All his life	Nov., 1924	March, 1925	6 × 5 cm.	4 months	Carcin tion ;
6	T. R.	66	Groom	All his life	Oct., 1924	July, 1925	Not known	8 months	Carcin no pr

pharyngeal, the palatal and tonsillar branches of the facial, the lingual, and the suprahyoid branches of the superior thyroid vessels.

Onset and Course.—In these cases growth appears to have commenced in this gland and steadily increased in size, maintaining until late its regularity. Growth is centrifugal, and in its cranial course gradually fills the space between the upper third of the sternomastoid laterally and the ascending ramus of the mandible medially. These two structures tend to limit its spread, and their constant movement aids the production of the ovoid form which these tumours maintain until a late stage in their development. Situated on the jugular vein, and beneath the deep fascia, it is free to extend laterally beneath the sternomastoid, medially towards the pharynx, and downwards into the neck, its upward spread chiefly running along the plane of the great vessels, the mastoid process and auditory meatus forming a strong buttress in the path of its more superficial route. Growth is no doubt directed mainly towards the softer structures of the pharynx, spread in this direction occurring beneath and being directed by the attachment of the deep fascia and the stylomandibular ligament to the mandible.

Site of Origin of the Primary Focus.—In only one of these cases was necropsy possible (*Case* 3), and in this case the primary focus was found in the pyriform fossa. In view of the size of the minute ulcer and small papilloma found, it is not surprising that during life this focus was overlooked, and no doubt at the time of examination the papilloma was not present; without it, even at careful necropsy, the ulcer might have been overlooked. In *Case* 1 the clinical findings point definitely to the site of origin being in the posterior nares, and it is seen that there was only a detectable roughening at this site at the first examination, and it was not until six months later that the focus made itself definitely apparent.

PRIMARY MALIGNANT TUMOUR OF THE NECK.

Histological Picture	Ultimate Fate	Primary Focus
Carcinoma, squamous in type; no prickle cells; no cell nests	Died July, 1925	Posterior nares. Clinical proof. No post-mortem
Carcinoma, squamous in type; no cell nests; prickle cells present	Died April, 1925	No primary focus found clinically. No post-mortem
Carcinoma, squamous-celled; no cell nests; prickle cells present	Died Nov., 1925	Post-mortem performed. Primary focus, sinus pyriformis
Carcinoma, squamous-celled; cell nests; prickle cells present	Died May, 1925	No post-mortem. No primary focus found clinically
Carcinoma, squamous in type; no keratinization; prickle cells present	Alive and well	No primary focus found clinically
Carcinoma, squamous in type; no cell nests; no prickle cells	Alive	No primary focus found clinically

Necropsy was unfortunately unavailable in the remaining four cases. It may be emphasized here that it is the difficulty of obtaining post-mortem examination that has in the past led to the diagnosis of branchiogenetic carcinoma merely on the score of absence of a primary focus in life.

The Occupational Similarity.—It is interesting to note that the cases of this group occur either in miners or men who have worked with horses all their lives. The curious occupational similarity was so striking that a search was made in the hospital cancer and surgical register for further evidence of cases giving a similar clinical history and a corresponding anatomical site of growth. Three cases were found; one occurred in an elderly miner, one in a young carter, and the third in a man who had worked with horses all his life, but prior to his death was occupied as an engineer.

The question immediately arises: Can there be any possible connection between the environment of these patients and their disease? The miner works in a dust-laden atmosphere, and those who have groomed a horse will know that the groom also is subjected to continual chronic irritation of the respiratory tract. In both, as in all of us, the pyriform fossa can form the site of lodgement of a small foreign body, and is continually subjected to the stagnation of particles of swallowed salivary or nasal secretion. But of the two it is essentially the groom, who is in the habit of chewing either a wisp of straw or hay, or a few of the oats from his horse's feed, who is, in consequence, more liable to a lesion of this area or of the tonsil. It is feasible, therefore, to suggest that, in those occupations in which a certain anatomical tract is subjected to the continual risk of trauma, such a site must be regarded as a possible mode of entry by a common causal agent, either at the time of its production by actual inoculation or by a subsequent infection from the inspired or swallowed content of a pollen or dust-laden atmosphere.

Treatment.—With one exception these particular cases when first seen had received no treatment other than medical. In the exception a surgical removal had been performed at another hospital, and three months later this patient was admitted with a fair-sized recurrence at the operation site.

It may be stated now that the great majority of recorded cases of surgical removal are uniformly disappointing; recurrence is generally rapid and fatal, whether a local or drastic operation has been performed. The reason for these poor results appears to be due to two main facts: (1) The difficulty of early diagnosis owing to the site of the tumour; (2) The presence of a primary focus acting as a neoplastic cell depôt.

An enlargement of this jugulo-digastric gland is most commonly due to the chronic infection, and the diagnosis of malignancy will only be suspected when the gland loses its mobility by its attachment to the great vessels upon which it lies. At this stage surgical removal without damage to the great vessels is difficult, but still possible. At a late stage the operation must be abandoned or the vessels resected with the growth. If either of these surgical feats is accomplished—and they have been in the past—it is evident that a large traumatized area is left behind, leading into which are the severed conduits of the primary focus which is still intact. Recurrence appears to be inevitable and it is rapid, since all fascial barriers have been removed and an excellent breeding ground for neoplastic cells is produced.

In the cases recorded here the question of operation did not occur; they were all hopelessly inoperable at the time when first seen, and the only possible course to pursue was either to refuse treatment, allowing them to die of their inevitably fatal disease, or to employ some form of X-ray or radiotherapeutic measure. The method chosen was radium.

In the treatment of these particular cases one had, therefore, to contend not only with the primary focus, but with a large mass of secondary growth. This volume of the tumour is extremely important from the point of radium therapy, as it is upon volume that the hope of correct dosage at present depends. The reason that this is emphasized is to draw attention, as has previously been shown, to the depth of the tumour and its liability to encroach upon the pharynx and travel towards the skull along the great vessels, so that always there is a tendency to under-estimation of the true size of the growth.

In examining the results of treatment in these cases it is seen that one is still alive and well, and that in another the original tumour-bearing site was apparently free from growth at the time of his death. With regard to the case that is still alive, only ten months have elapsed from the date of treatment, so that it is too early yet to draw definite conclusions; but it is significant that his tumour was the smallest of the series, having a surface measurement of 6 × 5 cm., giving a volume of roughly 150 c.c. The dose given was 200 mgrm. for twenty-four hours—an equivalent of over 1 mgrm. per 1 c.c. In the other case two applications were given to a tumour measuring 9 × 6 cm., whose volume was roughly 400 c.c. The first dose was 225 mgrm. for twenty-four hours, and the second 300 mgrm. for thirty hours, making a total of over 1 mgrm. per c.c.

Of the three other cases, in one the tumour was 12 × 11 cm. and too far advanced for any favourable result; the other two improved, and the growth

diminished in size for a time, but their dose appears totally inadequate, being roughly 1 mgrm. to 2 c.c. of tissue. In the future, should it be considered advisable to employ radium as a method of treatment, it appears that the best course to pursue is to compute as far as possible the correct volume of the malignant growth, and, owing to the depth of the tumour, to err on the generous rather than the meagre side; having obtained this volume, a total dose should be devised which approximates as far as possible to 1 mgrm. per c.c. for twenty-four hours. There are two methods of achieving this result: (1) By one or more doses using large tubes with a high radium content, the doses being repeated at relatively short intervals until the total amount is given; (2) The employment of small tubes of low radium content, leaving them in for a continual exposure of several days. Whichever method is used, it is essential to avoid normal tissue destruction and normal or tumour tissue necrosis—by screening to obviate the beta rays—and to produce uniform radiation to the deepest portion of the tumour that the anatomical structures will allow. Of the two methods outlined, the small needles and the long exposures appear to be the more suitable.

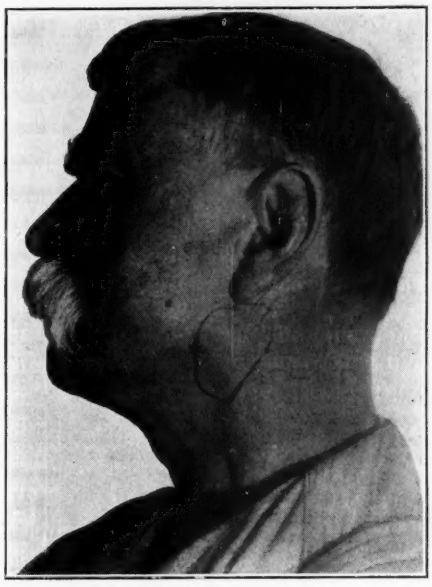

FIG. 177.—Carcinoma of the lower pole of the parotid gland. (From a case under the care of Mr. Gordon-Taylor.)

Diagnosis.—The diagnosis of these tumours rests principally upon their anatomical site and their consistency. The solid tumours that may occur at the same site are the primary endothelioma of lymph glands when it is of the single type, and the endothelioma of branchiogenetic origin; microscopic sections only will prove diagnosis. The solid tumours occurring near the site, in addition to the above-mentioned tumours, are the endothelioma and especially the carcinoma commencing in the lower pole of the parotid gland (*Fig.* 177); but these growths are always at a higher level, more superficial, and spread in the parotid substance and cheek, as well as the post-ramal recess. Tumours of the carotid body are situated at a lower level and tend to be globular and early irregular in outline; their histological picture is definite.

All the above tumours are extremely rare. The growth most likely to give difficulty in diagnosis is the branchiogenetic carcinoma arising in a pre-existing cyst (*Fig.* 178), since its anatomical site and histological picture are somewhat similar; but it is again emphasized that the tumour is essentially cystic in some parts, and that it occurs at a lower and more median level in the neck.[4]

Early diagnosis will always be difficult, but the presence of a unilateral painless tumour at the site of the jugulo-digastric gland in an elderly man must be regarded with grave suspicion.

SUMMARY.

Six cases only have been recorded, and from so small a number it is unsound to draw too definite conclusions. It is submitted that these tumours form a definite etiological, pathological, and anatomical group :—

1. That they occur in those subjected to chronic irritation of the upper respiratory tract—in miners and workers amongst horses.

2. That they are derived from a primary focus in the region drained by the jugulo-digastric gland.

3. That their treatment is essentially by methods other than surgical, and at the present time, in the absence of a better therapeutic measure, radium is the method of choice.

4. That they are constantly being diagnosed erroneously as branchiogenetic in origin, and this has tended to bring the possibility of carcinoma arising in branchial remnants into disrepute.

5. That they may throw some light upon the pathway by which the causal agent of certain cases of malignant disease, and certain of the granulomas, gain an entry to the body.

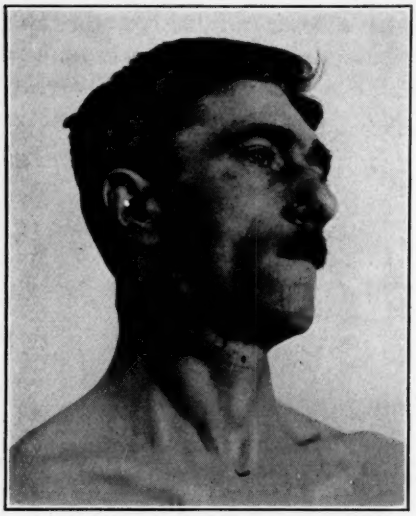

FIG. 178.—Showing the site of a branchial cyst. (From a case under the care of Mr Shorney Webb.) X marks the angle of the jaw. The upper dot in the middle line is opposite the centre of the body of the hyoid bone. The lower dot is opposite the centre of the notch of the thyroid cartilage. Note the lower level of the site of the cyst.

I have to thank the Members of the Radium Committee of the Middlesex Hospital for their kind permission to publish these cases, and for their helpful criticism in the completion of this paper. Particularly are my very grateful thanks due to Mr. Sampson Handley for his helpful advice and criticism.

My thanks are also due to Miss D. Clephan for photographing and carrying out the diagrams of these cases to my design.

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[2] EDMUND, ARTHUR, *Choyce's System of Surgery*, iii, 206.
[3] JOHNSON, RAYMOND, *Ibid.*, i, 559.
[4] BAILEY, HAMILTON, "The Clinical Aspect of Branchial Cysts", *Brit. Jour. Surg.*, 1923, April, 565.
[5] HANDLEY, SAMPSON, "The Encirclement Method of Using Buried Radium Tubes", *Brit. Jour. of Radiol.*, 1925, Nov., 422.

THE IMPORTANCE OF TOXÆMIA DUE TO ANAEROBIC ORGANISMS IN INTESTINAL OBSTRUCTION AND PERITONITIS.*

A clinical and experimental investigation, with the results of treatment with the appropriate antiserum.

By B. W. WILLIAMS, London.

INTRODUCTION.

The similarity between the general clinical manifestations of cases of acute peritonitis and acute obstruction is, I think, widely admitted. In most fatal cases of acute peritonitis paralytic obstruction of the intestine develops secondarily, and it is this obstruction which produces the characteristic clinical aspect in the later stages. Taking the features common to the two conditions, though there is variation in individual intensity, collectively they bear a striking resemblance to the clinical features of the toxæmia associated with gas gangrene.

In the report of the Committee upon Anaerobic Bacteria and Infections published by the Medical Research Council the clinical aspect of anaerobic toxæmia resulting from war wounds is described as follows: "Vomiting is frequent . . . the pulse becomes rapid and is finally uncountable . . . the extremities are cold and blue, and the temperature falls. . . . It is a remarkable fact that the mind remains acute even to the end. In the terminal stages some degree of general icterus may be present".[1]

It is interesting to compare this with the descriptions in a standard textbook of the clinical aspect of late cases of intestinal obstruction, or peritonitis. In a well-known manual of surgery the typical appearance of a late case of intestinal obstruction is described as follows: "The face is pale . . . the pulse rapid and feeble . . . the hands and feet, the tips of the nose and ears become cold. . . . He usually remains conscious to the end, and may fail to realize the gravity of his condition".[2] In the same book the following account is given of the terminal stages of acute peritonitis: "The face is pale and drawn, and assumes an anxious expression. The eyes are clear and bright, and mental faculties are often abnormally alert. . . . The face and extremities become cold and cyanosed . . . the pulse becomes small and running. . . . As a rule the patient remains conscious to the last, sometimes ceasing to complain, or even expressing himself as feeling better".[3]

The variation in the local conditions in the different diseases tends to mask the general resemblance; but if these local signs are excluded, the common features present a symptom-complex which is striking and peculiar when compared with almost any other acute toxæmia, such as that of

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streptococcal septicæmia, cellulitis, typhoid, diphtheria, pneumonia, or uræmia. Both in acute intestinal obstruction and acute peritonitis the patient is suffering from an acute toxæmia, and the clinical resemblance in the two diseases suggests the probability that the toxæmia originates in the same way in each.

In acute intestinal obstruction dehydration and a loss of salts by vomiting occur, but cannot be considered adequate cause for the toxæmia. The only remaining obvious source of toxin is the contents of the obstructed intestine. The toxicity of the intestinal contents has long been recognized by surgeons, as witnessed by the importance which is attached to obtaining immediate drainage of the obstructed bowel, rather than dealing with the cause of the obstruction, as a primary object of operation.

In acute peritonitis the type of toxæmia under discussion appears only in the later stages, in association with paralytic obstruction of the intestine. Here, again, the importance of obtaining drainage of the paralysed gut is widely, though not generally, recognized. Many procedures have been advocated with this object. The difficulties of draining bowel which has lost the power of contraction are, however, very great, and no method as yet devised is entirely satisfactory. This problem will be referred to again later.

The foregoing considerations suggested to me the possibility that the peculiar toxæmia described might result from the abnormal proliferation in the obstructed intestine of anaerobic organisms.

THE NORMAL DISTRIBUTION OF ANAEROBES IN THE INTESTINAL TRACT.

It is first necessary to consider the normal distribution of anaerobes in the intestine. Many anaerobes have been found in the human intestine. *B. sporogenes*, *B. welchii*, *B. tetani*, *B. hystolyticus*, and many others have been isolated. Whether they are present as a natural flora or as passengers in the food is immaterial to the problem under discussion. Of those organisms which are known to produce toxins, *B. welchii* is by far the most abundant and most constant. It is an organism which requires for its growth and the production of toxin an anaerobic medium, which should be neutral or slightly alkaline. In acid medium the toxin is destroyed,[4] and *B. welchii* would be rapidly outgrown by other organisms. Herter[5] has shown that in the greater part of the small intestine the conditions are anaerobic, and the medium is alkaline, while in the large intestine the medium becomes acid, and in spite of the immense proliferation of organisms of all sorts in this portion of the gut, *B. welchii* is frequently absent from cultures of normal fæces.[6] More recently Dudgeon[7] found *B. welchii* present in only 35 per cent of 200 specimens of fæces from human cases.

The lower part of the small intestine thus appears to be the only part of the bowel suitable for proliferation of *B. welchii* and the formation of toxin. The passage of material through the small intestine is, however, extremely rapid. Normally, a barium meal passes through to the cæcum in three to four hours, and it is extremely rare to see any prolonged delay in the small intestine. During this period the reaction changes from acid to strongly

alkaline, back to neutral or slightly acid in the terminal ileum, so for only a fraction of that time is the reaction suitable. When the small bowel becomes obstructed or paralysed, however, ideal conditions exist for the proliferation of *B. welchii* and the formation of toxin.

In pyloric obstruction the acid reaction in the stomach and aerobic conditions would prevent proliferation and destroy toxin, and clinically pyloric obstruction gives rise to no acute toxæmia. Similarly in obstruction of the large intestine: so long as stagnation is confined to the large intestine, apart from possible differences of absorption, the acid reaction of the contents would destroy *B. welchii* toxin. Clinically it is a matter of common observation that constipation or intestinal obstruction gives rise to no acute toxæmia until the stagnation extends back into the small bowel, either by incompetence of the ileocæcal valve, or by increased difficulty of emptying the small intestine against increased pressure. It is therefore suggested that it is the extension of stagnation into the small intestine which marks the transition of chronic to acute intestinal obstruction in cases where the organic obstruction is situated in the large bowel. Broadly speaking, organic obstruction arising primarily in the small bowel is acute from the onset; that arising primarily in the large bowel may be chronic at first.

On this hypothesis there should therefore be a great proliferation of *B. welchii* in the small intestine of patients suffering from acute obstruction. In the later stages of the disease these organisms would be found in increased numbers higher up the intestines, and eventually in the vomit, though in the stomach proliferation would be checked actually by the acid reaction, and toxin destroyed. These considerations led me first to examine the vomit and the contents of the small intestine, in cases of acute obstruction and of peritonitis, for evidence of proliferation of *B. welchii*.

EVIDENCE OF THE PROLIFERATION OF B. WELCHII IN THE OBSTRUCTED OR PARALYSED SMALL INTESTINE.

Methods of Examination of Vomit and Small Intestine Contents.—As soon as possible after the material was obtained, films were made on an area of 1 square inch of slide, using one standard platinum loop of 2-mm. diameter. These films were stained by Gram's method, and give a rough idea of the relative numbers of organisms present.

Aerobic cultures were made by diluting one standard loopful of material in 2 c.c. of normal saline, and spreading one standard loopful of the diluted material on an agar plate of 3 in. diameter.

Anaerobic cultures were made by inoculating one standard loopful of material into tubes of Robertson's meat medium and milk, which had been previously heated for half an hour in boiling water. These were placed in a vacuum jar, which was exhausted to a negative pressure of 30 in. of mercury with a water-pump, and washed out with hydrogen at least three times. This method gave satisfactory anaerobic conditions, and surface colonies could be readily grown on solid media.

The identification of *B. welchii* depended upon: (1) The presence of organisms morphologically resembling *B. welchii* in the stained films and in

meat medium cultures; (2) The formation of storm-clot in milk; (3) The results of mouse inoculation, using pure antisera for *B. welchii*, *B. œdematiens*, and *Vibrion septique* as controls.

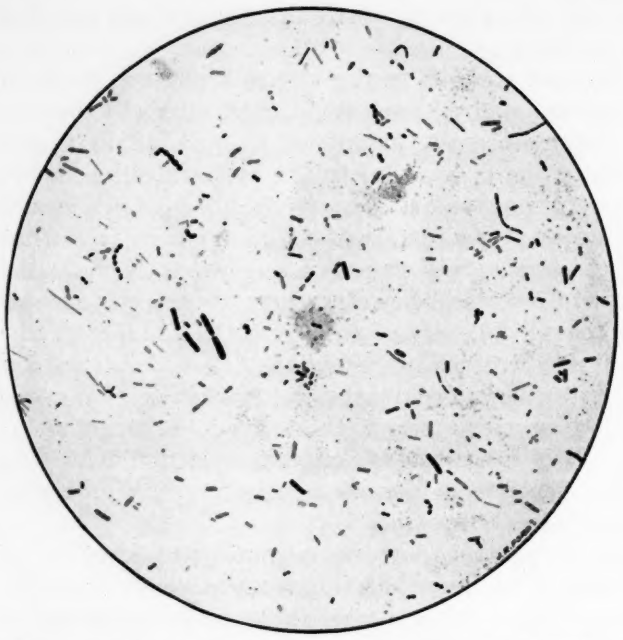

FIG. 179.—Standard film of vomit from a case of peritonitis.

Nearly all the specimens were, of course, heavily infected with other organisms. Amongst the aerobes, streptococci and coliforms were the most abundant, while amongst the anaerobes, *B. sporogenes* was usually present, but not apparently abundant in early cultures. Once mouse inoculation suggested the presence of *Vibrion septique*, but I failed to isolate it from the other organisms, and it apparently died out on sub-culture.

Examination of Vomit.—In vomit obtained from cases of acute obstruction, *B. welchii* was found in 11 out of 12 specimens. In vomit obtained from advanced cases of peritonitis, *B. welchii* was found in 19 out of 20 specimens. Some of the films prepared from fresh vomit showed large numbers of organisms indistinguishable from *B. welchii* (*Figs.* 179, 180), while in others these organisms were comparatively few. The numbers present in the original films did not, however, bear any constant relation to the severity of the case. This disparity may have been due to dilution of the intestinal material in the stomach, either by secretion

FIG. 180.—Standard film of vomit from a case of obstruction.

or by fluid taken by mouth. The vomit was usually obtained immediately after admission, before operation. As a control, vomit was examined from two cases of pyloric obstruction, one simple and one malignant, and from three cases in which there was no intestinal obstruction. In none of these was any evidence found of the presence of *B. welchii*.

Twelve test meals performed for various conditions were also examined, and in none were any organisms found resembling *B. welchii* on the stained films; in one test meal a positive result was obtained on culture.

FIG. 181.—Standard film of small-intestine contents from a case of peritonitis.

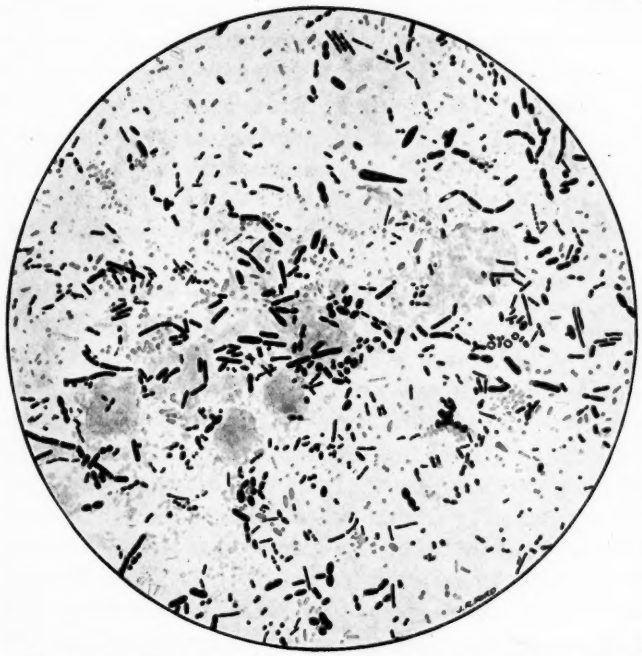

FIG. 182.—Standard film of small-intestine contents from a case of obstruction.

Examination of Small Intestine Contents.—The contents of the small intestine obtained post mortem from four cases of peritonitis, and one of acute obstruction, all showed large numbers of *B. welchii*.

Six specimens of small-intestine contents were obtained during life from human cases of intestinal obstruction and peritonitis. Four were cases in which jejunostomy had been performed, one for volvulus, one for obstruction by bands, two for peritonitis; the fifth was a case in which 2 feet of small intestine were resected for volvulus, and the sixth a duodenal fistula and

peritonitis from acute cholecystitis. In all of these specimens enormous numbers of *B. welchii* were present (*Figs*, 181, 182). *B. welchii* was equally abundant in the small intestines of dogs in which acute obstruction and peritonitis had been produced in an experiment which will be described later.

As a control I was only able to obtain intestinal contents from one human case, killed in a street accident. Food was last taken four hours before death, and the material examined six hours after death. Very few organisms could be found in the stained film preparations of the upper part of the small intestine, but *B. welchii* was cultivated from the middle and lower part of the jejunum. In the lower ileum (*Fig.* 183) a fair number of *B. welchii* were seen, though the numbers were very small when compared with those present in the cases of intestinal obstruction and peritonitis. This corresponds with the results obtained in human beings by A. Goldman,[8] and with observations made on the intestinal flora of normal dogs by Torrey,[9] and confirmed in four normal dogs by myself.

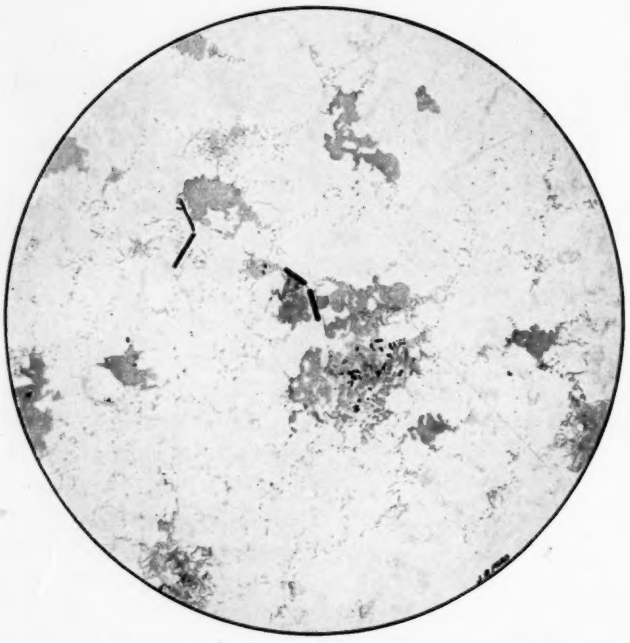

FIG. 183.—Standard film of contents of normal terminal ileum. A selected field showing organisms resembling *B. welchii*.

CONCLUSIONS AS TO PROLIFERATION OF B. WELCHII IN THE OBSTRUCTED OR PARALYSED SMALL INTESTINE.—*B. welchii* is present in enormously increased numbers in the small intestine contents and in vomited intestinal contents in cases where there is obstruction, whether organic or paralytic. There is at the same time an immense increase in other bacteria, but *B. welchii* is the only organism known to produce a powerful exotoxin. The morphological appearances of *B. welchii* are variable in the extreme, and I do not think it possible to enumerate individual anaerobic organisms with any degree of certainty on stained films. In consequence of this, no attempt was made to count the relative numbers in the original films.

It is, of course, improbable that great proliferation of *B. welchii* would take place under the conditions in the small intestine without formation of toxin; but one knows that with *in vitro* cultures toxin is not always easy to demonstrate in spite of active growth, and apart from its toxin *B. welchii* is a very innocuous organism. It is essential therefore that some evidence should be found for the presence of toxin in the contents of the obstructed intestine.

EVIDENCE OF THE PRESENCE OF B. WELCHII TOXIN IN THE CONTENTS OF OBSTRUCTED OR PARALYSED INTESTINE.

Attempts were made to demonstrate the presence of *B. welchii* toxin in the vomit and contents of the small intestine of advanced human cases, and, in view of the small number of human cases available, experimentally in dogs.

Methods.—The presence of *B. welchii* toxin in fluids can be demonstrated only by animal inoculation. The effects of the bacteria-free fluid mixed with normal horse-serum are compared with the effects of bacteria-free fluid mixed with *B. welchii* antitoxic serum. The recognized tests are: (1) Lethal effects in mice and guinea-pigs; (2) Intradermic reactions in guinea-pigs; (3) Lethal effects, or microscopical changes produced in the pectoral muscles, in pigeons. The presence of bile, irritant substances, and wide variations in reaction render the last two methods unsuitable for intestinal or vomited material, as I found that controls frequently exhibited doubtful changes. Consequently the first method only was used. Mice were selected, in order that, with a limited amount of material, the number of animals used might be as large as possible.

The difficulties of demonstrating the presence of toxin in vomit or intestinal contents are, however, very great. The toxin is extremely labile, and is rapidly destroyed by heat, or by slight variation in acidity.[4] Even at laboratory temperature, toxin prepared *in vitro* may disappear in a few hours, if the reaction is unsuitable. The toxin is non-dialysable, and a considerable proportion is lost, even by rapid passage through bacteriological filters, so that sterilization is best effected by rapid centrifugalization. This method, however, proved to be unreliable with the centrifuge available, when applied to vomit or intestinal contents, on account of the presence of large amounts of mucus. In order therefore to obtain a bacteria-free inoculum, the material had to be passed through a Berkefeld candle (N. & V.), although it was known that a large proportion of any toxin present would be lost by this method. Filtration was extremely slow, even with preliminary centrifugalization, and as a rule occupied twenty-four hours. Filtrations were conducted in an atmosphere of hydrogen in the cold, in the hope of checking destruction of toxins.

Wide variations were found in reaction in different specimens; in the case of vomit the reaction was nearly always strongly acid, and accordingly unsuitable for intravenous inoculation. For the sake of uniformity, therefore, intramuscular inoculation was always used, though I believe that it usually requires more toxin and takes longer to obtain lethal results.

Examination of Vomit.—It will be seen from the foregoing reasons that it would be very improbable that any toxin could be demonstrated in vomit, and, in fact, of seven specimens of vomit tested in this way, all from extreme cases of peritonitis or acute obstruction, only from one were lethal effects obtained in mice. This vomit was from a case of general peritonitis in the terminal stages, and was neutral in reaction. Two mice which had received 0·75 c.c. of vomit filtrate in 0·25 c.c. normal horse-serum died within five days of inoculation. Two mice which had received protective *B. welchii* antitoxic serum and vomit filtrate in similar amounts were unaffected.

Examination of Intestinal Contents.—I have found great difficulty in obtaining material from the small intestine of human cases of acute obstruction or of peritonitis. Material obtained post mortem would be open to the obvious criticism that toxins might be formed after death.

Jejunostomy and ileostomy are not frequently performed on these cases at St. Thomas's Hospital, and during the last three years I have only obtained material from four cases, and only in two was there sufficient material for filtration. One was a case of small-bowel obstruction by bands, and one a case of peritonitis due to appendicitis. The technique adopted was the same as is described below in the examination of the intestinal contents of dogs, and the results of mouse inoculation are represented in *Table I.*

In view of the difficulty of obtaining adequate human material, it was decided to examine the small-bowel contents of dogs in which acute obstruction or peritonitis was produced experimentally. In three dogs peritonitis was produced as follows: The abdomen was opened under anæsthesia, the base of the appendix was ligatured so as to strangulate its blood-supply, and the abdomen closed. Later, on the appearance of definite abdominal distention, the animal was again anæsthetized, and, with continuous subcutaneous saline infusion, was kept alive as long as possible under anæsthesia, while the thoracic duct was drained in the neck in connection with an experiment which will be mentioned later (*see* p. 306). In two dogs acute obstruction was produced by ligaturing the ileum 3 in. above the ileocæcal valve, without interfering with the blood-supply. The remainder of the experiment was carried out as has been described above. Blood cultures were made at intervals during the later stages in connection with experiments mentioned on p. 306.

Immediately after the death of the animal the contents of the stomach and small intestine were examined. Stained films and cultures were made according to the technique already described. In each case enormous numbers of *B. welchii* were present in the contents of the small intestine. A similar flora, but in much smaller numbers, was found in the contents of the stomach. The smaller number of organisms in the contents of the stomach can only have been produced by dilution of the intestinal material by secretion.

The contents of the small intestine were then mixed with an equal volume of ice-cold normal saline, and were passed through a Berkefeld candle in an atmosphere of hydrogen in the ice-chest. Filtration was always slow and difficult, and took eighteen to twenty-four hours, even after dilution with saline. Cultural controls for sterility were put up, and mouse inoculation was then carried out.

Protected mice received 0·75 c.c. of filtrate and 0·25 c.c. of *B. welchii* antitoxic serum intramuscularly. Unprotected mice received 0·75 c.c. of filtrate and 0·25 c.c. of normal horse-serum. The mice were kept under observation for six days after inoculation, and those which died were examined for the possibility of infection with *B. welchii*, as opposed to toxæmia, by heart-blood culture, as soon after death as possible. All these cultures were negative as regards *B. welchii*, though occasionally streptococci and coliforms were found. The results of these inoculations are represented in the following table :—

Table I.—Results of Inoculation of Intestinal Contents in Mice, indicating the Presence of B. Welchii Toxin.

Source of Intestinal Contents	Duration of Condition	Unprotected Mice		Protected Mice	
		Total	Died	Total	Died
Human case, peritonitis ..	7 days	10	5	5	0
Dog, peritonitis, 1 ..	83 hours	4	4	2	0
,, ,, 2 ..	65 hours	6	6	2	0
,, ,, 3 ..	78 hours	6	4	4	0
Human case, obstruction ..	3 days	12	2	6	0
Dog, obstruction, 1 ..	56 hours	8	7	4	0
,, ,, 2 ..	51 hours	8	0	4	0
Total mice used ..	..	54	28	27	0

The total number of mice used is, of course, small, and was limited by the difficulty of getting a larger amount of material through the filters in reasonable time. For the same reason the number of control mice was reduced. The material obtained from the second dog in the acute obstruction experiment was particularly troublesome in filtration, which may explain the entirely negative result obtained on this occasion.

The number of experiments is also small, but the procedure is a severe one, and I did not feel justified in further repetitions. The difficulties of working with *B. welchii* toxin at any time, and the special difficulties encountered in material which is full of mucus and of varying reactions, justify the deduction that a considerable concentration of *B. welchii* toxin is present in the small intestine in cases of peritonitis and obstruction.

As a control, the contents of normal human ileum, obtained six hours after death, the last meal having been taken four hours before death, were examined by the same method. Ths filtrate produced no ill effects in mice, which agrees with the findings of Davis and Stone.[10]

In view of the absence of clinical toxæmia in obstruction confined to the large intestine, there should be no toxin in the contents of obstructed large intestine. Material was obtained from three patients suffering from obstruction due to malignant disease, on whom colostomy was performed, and the filtrate inoculated into mice. Filtration was much more rapid than in the case of small intestine contents, and no dilution with saline was necessary. Large quantities also were available. With the three specimens, 16, 25, and 25 unprotected mice were inoculated. No serious ill effects were observed and there were no deaths. There was, therefore, no toxic substance found in the contents of the large intestine, even in the presence of obstruction, though stained film preparations of the same material showed large numbers of *B. welchii*.

Conclusions as to Presence of Toxin of B. Welchii in the Obstructed or Paralysed Intestine.—(1) In one human case of peritonitis there was questionable evidence of the presence of *B. welchii* toxin in the vomit. (2) In one human case of peritonitis and one of acute obstruction

there was evidence of the presence of *B. welchii* toxin in the contents of the obstructed small intestine. (3) In the small-intestinal contents of 5 dogs in which obstruction or peritonitis had been produced experimentally, the presence of *B. welchii* toxin was demonstrated. Of 32 inoculated mice, lethal effects were obtained in 21, and 16 control mice that were protected with *B. welchii* antitoxin were unaffected. (4) *B. welchii* toxin was the only toxin found which was lethal to mice. (5) Contents of normal human ileum were found to be non-toxic in one case. (6) Contents of obstructed large intestine were found to be non-toxic in 3 human cases.

EVIDENCE OF ABSORPTION OF B. WELCHII TOXIN FROM OBSTRUCTED OR PARALYSED INTESTINE.

It only remains to be shown that *B. welchii* toxin is absorbed from the intestine and is responsible for the toxæmia. That cases suffering from acute intestinal obstruction and from peritonitis show clinically a toxæmia, and that the toxæmia bears a striking resemblance to that resulting from gas gangrene infections, may be taken as evidence of absorption. Peculiar though the toxæmia is in many respects, still it is possible that the clinical features might be produced in other ways.

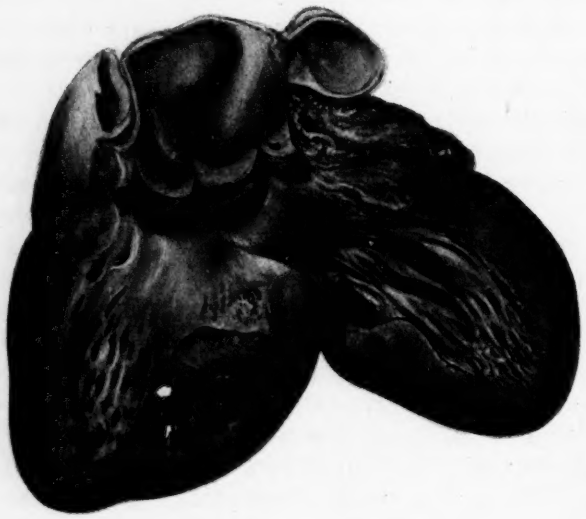

FIG. 184.—Heart showing hæmolytic staining. From a case of pneumococcal peritonitis.

One of the most marked properties of *B. welchii* toxin is its hæmolytic power, and cases of acute obstruction and of peritonitis were examined for evidence of hæmolysis. The success of the clinical trial to be described later rather interfered with this part of the investigation. Anæmia and slight general icterus are well-known clinical features of severe cases which survive for some time, but I have only recently begun to perform definite blood examinations. Hæmolytic staining of the intima of the great vessels and heart is a well-known post-mortem finding in cases of peritonitis, and was frequently found. Hæmolytic staining post mortem in cases of acute obstruction also is not infrequent. I have found it present in seven out of eleven cases, and in some the staining was very marked. It is easy, and I believe usual, to postulate the presence of a hæmolytic streptococcus in these cases, though there is no other clinical or post-mortem evidence of its presence. Had a streptococcal septicæmia been responsible for the condition, one would have expected other manifestations, such as changes in the spleen, and metastatic foci. These were certainly not present in the cases referred to above. *Fig.* 184

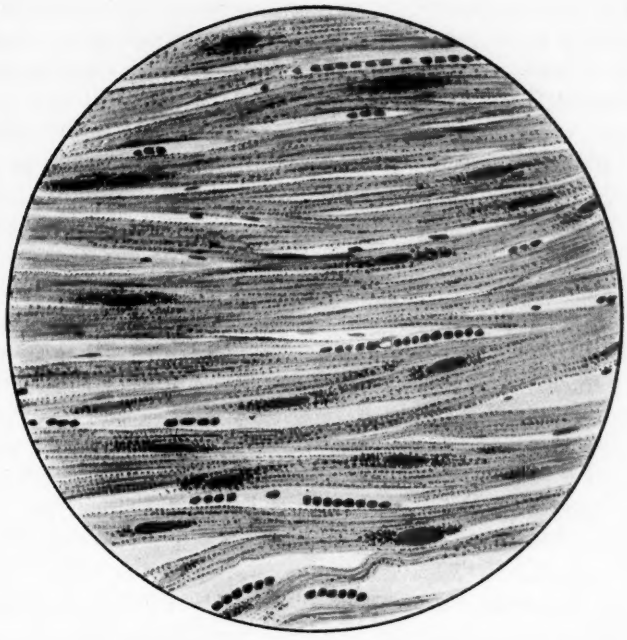

FIG. 185.—Cardiac muscle from fatal case of small-bowel obstruction, showing fine fatty degeneration.

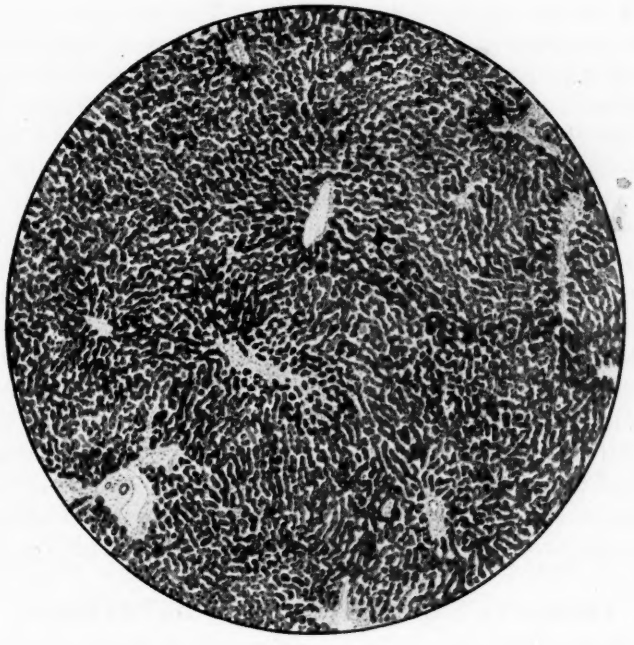

FIG. 186.—Liver from fatal case of small-bowel obstruction, showing fine fatty infiltration.

shows the heart from a fatal case of pneumococcal peritonitis in a girl 3 years old. The brilliant hæmolytic staining of the lining of the heart and great vessels seen here could not have been produced by the pneumococcus, which, though it forms methæmoglobin from blood *in vitro*, is not in the ordinary sense hæmolytic. This staining, however, may well have resulted from the absorption of *B. welchii* toxin from the stagnant contents of the paralysed small intestine, which was greatly distended. In the experiment already described, of the five dogs in which obstruction or peritonitis had been produced experimentally, four showed marked hæmolytic staining. Blood cultures were made in all of them within a few hours of death, and in only one was a positive result obtained; streptococci, coliforms, and *B. welchii* were grown.

Histologically, no very definite generalized changes are associated with *B. welchii* toxæmia. Kettle[11] in cases of gas gangrene found fatty degeneration in the liver, of no particular distribution, and in the heart muscle. Stone and Bernheim[12] found fine fatty degeneration in the liver of dogs with intestinal obstruction; and in the heart muscle and liver of eight human cases of peritonitis or obstruction, I have found similar changes in varying degrees (*Figs.* 185, 186). The changes recorded above are all compatible with, though not diagnostic of, a *B. welchii* toxæmia.

In view of the claims put forward recently by Costain[13] and Edwards,[14] that the mortality of peritonitis could be materially diminished by drainage of the thoracic duct in the neck, an attempt was made to examine the lymph obtained from dogs, in which peritonitis or obstruction had been produced experimentally, for the presence of *B. welchii* toxin. The finding of *B. welchii* toxin in the lymph of the thoracic duct would be absolute proof both of the formation and of the absorption of toxin. The results of this experiment were, however, entirely negative. This negative result must not be regarded as damaging to the hypothesis in any way. Substances of relatively small molecular dimensions, like bacterial toxins, are probably more rapidly absorbed by the blood-stream than by the lymph, as has been shown by Bolton[15] and others. Further, *B. welchii* toxin, though present, might not be demonstrable on account of dilution in the lymph. Mr. J. H. Mason, of the Wellcome Research Institute, assisted with this experiment, which will be reported more fully elsewhere.

Conclusions as to Absorption of B. Welchii Toxin from Obstructed or Paralysed Intestine.—(1) The clinical features of late cases of acute intestinal obstruction and of peritonitis are compatible with the absorption of *B. welchii* toxin. (2) Evidence of intravascular hæmolysis as shown by staining of the endothelium of the heart and great vessels, in the absence of evidence of infection with other known hæmolytic organisms, is strong corroboration of the absorption of *B. welchii* toxin. (3) Fine fatty degeneration in the heart and liver of patients dying of acute obstruction and of peritonitis is compatible with the absorption of *B. welchii* toxin.

THERAPEUTIC TEST OF THE HYPOTHESIS.

The final test of the hypothesis was the administration of *B. welchii* antitoxic serum in suitable cases of peritonitis and obstruction. If the hypothesis is correct, it should be possible, with the high titre antitoxic sera prepared by

Burroughs, Wellcome & Co., to combat the toxæmia and reduce the mortality of these diseases. As a method of proof, a therapeutic test is always the least reliable of criteria, as the number of cases must inevitably be too small to eliminate the possibility of chance success or failure. In medicine, however, the therapeutic test must finally be the most important.

Anti-gas-gangrene serum has, I find, been used therapeutically in France for some time in cases of 'gangrenous appendicitis'. The results of this treatment were summarized by D. G. Michel[16] in 1923, and appeared to indicate a considerable reduction of mortality. The serum used by the French authorities is a polyvalent serum containing antibodies for a variety of anaerobic organisms, but with a considerable proportion of *B. welchii* antitoxin. The theoretical basis of its use depended upon the finding of *B. welchii* in a large proportion of acutely inflamed appendices by Veillon and Zuber,[17] and more recently by Weinberg.[18] Many observers, however, agree that *B. welchii* is only found, in the vast majority of cases, in the immediate vicinity of the perforated appendix, as a contamination by bowel contents, rather than as the cause of appendicitis or peritonitis. On stained film preparations of peritoneal pus from cases of acute appendicitis a few organisms that resemble *B. welchii* may frequently be seen, and I have often obtained them on culture. Their numbers are, however, quite insufficient to account for the profound toxæmia even when there is a considerable area of peritonitis. Further, in cases of pneumococcal peritonitis, in so-called 'primary peritonitis', and in many severe cases of peritonitis arising from acute appendicitis, when there is no macroscopic perforation of the appendix, *B welchii* is entirely absent from the peritoneal infection. Yet in all cases which develop 'ileus', whatever the cause of the peritonitis, the clinical picture is the same in the late stages, and this is the clinical picture of an acute anaerobic toxæmia. Obviously the accidental presence of a few *B. welchii* in the peritoneum, as a result of intestinal contamination, cannot, in the majority of cases, account for the toxæmia, though when present it may be a contributory source of toxin. Anaerobic infections of the peritoneal cavity may occur occasionally, but were practically unknown during the late war, though the attention of many observers was concentrated upon the anaerobic organisms. It may be that the impossibility of accepting *B. welchii* as the causative organism of appendicitis and peritonitis accounts for the fact that the administration of 'anti-gas-gangrene' serum, as advised by the French surgeons, has not attracted more attention in this country. In view, however, of the high proportion of *B. welchii* antitoxin contained in the French anti-gas-gangrene serum, the success of its therapeutic employment in cases of appendicitis reported by Michel[16] may be cited in support of the hypothesis put forward in this paper, that the toxæmia in late cases of peritonitis is due to the absorption of *B. welchii* toxin from the stagnant contents of the small bowel.

THERAPEUTIC TEST OF ANTI-GAS-GANGRENE SERUM IN CASES OF PERITONITIS.

At first it was decided to use cases of acute appendicitis for the clinical trial, because: (1) A fairly large number of cases are available at St. Thomas's Hospital annually; (2) Cases which are 'going wrong' give fair clinical warning,

which can be easily recognized, and definite signs of intestinal paralysis develop in nearly all fatal cases; (3) The mortality is well established, and fairly constant between 5 and 10 per cent. At St. Thomas's Hospital for the five years 1919–23 the average mortality was 6·5 per cent in 1418 cases.[19] The mortality of a disease depends to a large extent upon local conditions, the habits of the population, practitioners and surgeons, and the transport and the hospital facilities available. The figures for St. Thomas's Hospital only are therefore used for comparison.

In a condition like acute appendicitis the total figures include a large majority of cases which never develop serious intestinal paralysis, in which the mortality is correspondingly low. It is, however, only by taking the total figures that individual differences of opinion can be avoided as to what constitutes local, spreading, or 'general' peritonitis.

It was decided to adminster serum to all cases in which clinically one would say that the chances of survival were 50 per cent or worse. This included: (1) Moribund cases of severe general peritonitis; (2) Streptococcal peritonitis, unlocalized, in children under 12 years; (3) Cases which, after appendicectomy, continued to show signs of advance of the disease, by rising pulse-rate, abdominal distention, and toxæmia. In order to increase the number of cases, Mr. P. H. Mitchiner, Assistant Surgeon, St. Thomas's Hospital; Mr. A. C. Halliwell, Resident Assistant Surgeon; and Mr. C. V. Patrick, Surgical Registrar, consented to co-operate with me in the administration of *B. welchii* antitoxin to suitable cases. The series consisted of 256 consecutive unselected cases, which were distributed as follows:—

Analysis of Cases used in the Therapeutic Test.

Surgeon	Period	Total Cases	Deaths
A. C. H. ..	June 1, 1925, to June 1, 1926	91	2
B. W. W. ..	July 1, 1924, to June 1, 1926	82	0
C. V. P. ..	July 1, 1925, to June 1, 1926	34	1
P. H. M. ..	July 1, 1925, to June 1, 1926	21	0
Cases not operated upon during acute stage		28	0

Of the total 256 cases, 18 of the most severe were treated with *B. welchii* antitoxin, and of these, 10 were children under 12 years of age. Further details of the cases treated are given in *Table II*. There were 3 deaths, giving a mortality of 1·17 per cent. During the year 1925 a control series of 111 cases, consecutive and unselected, at St. Thomas's Hospital, under other surgeons who were not using *B. welchii* antitoxin, showed a mortality of 6·3 per cent.

Details of fatal cases.—

Case No. 35.—A woman, age 68, with thirty-two hours' history of symptoms. At operation the appendix was found to have perforated. There was some spreading peritonitis. Appendicectomy was performed and the abdomen closed. With the exception of some abdominal distention, progress was satisfactory until the nineteenth day after operation, when the patient was got up. No serum had been administered. After being got up, the patient became worse, and developed œdema of the legs and considerable abdominal distention. The bowels were kept open with

Table II.—Cases of Appendicitis in which B. Welchii Antitoxin was Administered.

No. in Series	Age in Years	Duration of Disease before Operation	Operation	When Antitoxin First Administered	Result	Remarks
22	5	72 hours	Appendicectomy	At operation	Recovery	Unlocalized streptococcal peritonitis
24	11	120 hours	Drainage	At operation	Recovery	'General peritonitis', appeared moribund
29	58	72 hours	Appendicectomy	At operation	Recovery	'General peritonitis',very ill
30	7	48 hours	Appendicectomy	At operation	Recovery	Unlocalized peritonitis, streptococcal type
32	11	72 to 96 hours	Appendicectomy	4th day after operation	Recovery	Progressive toxæmia after operation
33	5	72 hours	Appendicectomy	At operation	Recovery	Unlocalized peritonitis
35	68	32 hours	Appendicectomy	4 weeks after operation	Died 4½ weeks after operation	Cardiac failure (*see note, p.* 308)
37	3	48 hours	Appendicectomy	6th day after operation	Recovery	Progressive toxæmia, etc., after operation
44	43	56 hours	Appendicectomy	4 weeks after operation	Recovery	Intermittent obstruction during convalescence
47	39	48 hours	Appendicectomy	4th day after operation	Recovery	Progressive toxæmia, etc., four-month pregnancy aborted
48	4½	24 hours	Appendicectomy	3rd day after operation	Recovery	Progressive toxæmia and distention after operation
58	46	36 hours	Appendicectomy	4th day after operation	Died 6th day after operation	Local streptococcal peritonitis (*see note, p.* 310)
60	29	42 hours	Appendicectomy	At operation	Recovery	Spreading peritonitis, severe toxæmia
61	10	48 hours	Appendicectomy	At operation	Recovery	Spreading peritonitis, severe toxæmia
62	8	72 hours	Appendicectomy	At operation	Recovery	Unlocalized peritonitis, streptococcal type
63	6½	64 hours	Appendicectomy	At operation	Recovery	Marked distention and toxæmia
71	50	56 hours	Appendicectomy	4th day after operation	Died 4th day after operation	(?) Pulmonary thrombosis (*see note, p,* 310)
72	51	24 hours	Appendicectomy	3rd day after operation	Recovery	Progressive distention and toxæmia after operation

Total cases treated, 18; died, 3; children under 12 years old, 10.

enemata, but the patient sank and died thirty-one days after operation. *B. welchii* antitoxin was administered during the terminal stages.

POST-MORTEM EXAMINATION.—There were no signs of peritonitis. There was slight general distention of the intestine, but no mechanical obstruction could be found. The heart was small, brown, and soft. Death was reported to be due to cardiac failure.

Case No. 58.—A healthy policeman, age 46, with thirty-six hours' history of symptoms. At operation a perforated appendix was found, with spreading peritonitis. Two doses of castor oil had been administered before admission to hospital. On the third day after operation the abdomen was distended, he was slightly jaundiced, very restless and ill, with a pulse of 110. Pituitary and enemata failed to relieve the condition. On the fourth day the pulse was 120, the jaundice more marked, and the general condition worse. Fifty c.c. of anti-gas-gangrene serum were given intramuscularly. On the fifth day he appeared much better, distention was less, jaundice disappearing, and the pulse had dropped to 100. During the afternoon, however, he became rapidly worse ; distention and restlessness returned, and by 8 p.m. he was 'wandering'. A further 50 c.c. of anti-gas-gangrene serum was then administered. There was no improvement, and the patient died fourteen hours later.

POST-MORTEM EXAMINATION.—There was a very little pus about the right iliac fossa. The whole of the small intestine, except the last 6 in. of ileum, was grossly distended. There was very marked hæmolytic staining of the heart and great vessels, and slight jaundice of the sclerotics.

Case No. 71.—A woman, age 50, with fifty-six hours' history of symptoms. At operation a perforated appendix was found in an ill-defined pelvic mass, with some spreading peritonitis. Progress was satisfactory until three days after operation. No serum had been administered. On the third day the patient had an attack of cyanosis, pain in the chest, and cardiac failure. The abdomen became somewhat distended. There were two similar attacks during the next twenty-four hours, and after the third the patient became unconscious and died. *B. welchii* antitoxin was administered shortly before death. She vomited once after the first attack of pain, and coughed up blood-stained froth before death. The condition was suggestive of pulmonary thrombosis or so-called embolism. A post-mortem examination was refused.

In *Cases* Nos. 35 and 71 the administration of anti-gas-gangrene serum could not have any direct influence on the cause of death. They cannot, however, be excluded, because they would be included in any general mortality figures. *Case* No. 58 must be regarded as a failure. Serum treatment was not started as early as it might have been, and the patient was far gone before the second dose was administered ; but several cases which appeared to be quite as severe have recovered.

Clinical Effects of the Administration of B. Welchii Antitoxin in Cases of Peritonitis.—The clinical effects of the administration of serum were often striking and immediate. As a rule restlessness was greatly diminished or abolished, patients lost the 'acute consciousness', slept easily, and required little morphia to keep them quiet. Cyanosis, when present, disappeared, and in one case a definite icteric tinge faded rapidly.

In all successful cases the pulse-rate began to fall soon after the administration of serum. In cases which showed very marked toxæmia fall of pulse-rate sometimes did not occur for a day or two. One of the most striking effects was the influence of administration of serum on abdominal distention. Sometimes in the less severe cases distention disappeared and the bowels acted spontaneously within a few hours. In more severe cases, though the

bowels as a rule began to act spontaneously, in spite of previous failure of enemata and pituitary injections, distention subsided more gradually, and often did not completely disappear until convalescence was well established.

With the exception of the use of anti-gas-gangrene serum, these cases were, as a whole, treated by the ordinary methods in vogue at St. Thomas's Hospital. Cases seen during the first forty-eight hours of the disease were treated by appendicectomy. Excluding young children, patients seen after more than forty-eight hours after the onset of the disease were not operated upon until the acute attack had subsided, unless there were signs of spreading peritonitis or of the development of an abscess. In young children appendicectomy was always carried out as soon as possible. Drainage, other than that of the abdominal wall, was employed only when there was a definite abscess cavity, and when there was doubt about the suture line, or when the presence of concretions in the abdomen was suspected. In my own cases I did not employ injections of pituitary in the after-treatment.

THERAPEUTIC TEST OF ANTI-GAS-GANGRENE SERUM IN CASES OF ACUTE OBSTRUCTION.

I can find no record of anti-gas-gangrene serum having been used in this condition before. Cases of acute intestinal obstruction were not at first used in the therapeutic test—first, on account of the relatively small numbers

Table III.—Cases of Acute Obstruction Treated or Not Treated with B. Welchii Antitoxin.

Cause of Obstruction	Control Series 1919–23 Not Treated with B. Welchii Antitoxin		Test Series 1924–26. Treated with B. Welchii Antitoxin when necessary	
	Total No.	Deaths	Total No.	Deaths
Malignant obstruction with acute symptoms ..	28	11	10	1
Adhesions or bands ..	24	4	10	1
Internal strangulation ..	23	9	2	1
Intussusception with tumour	3	2	2	0
Volvulus, small intestine ..	3	2	1	1
Volvulus, large intestine ..	1	1	2	0
Inguinal hernia	44	6	11	0
Femoral hernia	66	11	13	1
Umbilical hernia	22	7	3	0
Totals	214	53	54	5

Mortality in control series, 24·8 per cent ; mortality in test series, 9·3 per cent.

available annually ; secondly, because it is impossible to predict before operation how any given case will behave. Patients sometimes die very suddenly after operation for no apparent reason, and successful cases seldom cause anxiety after relief of the organic obstruction.

In a series of 54 cases, however, *B. welchii* antitoxin was administered for therapeutic reasons in 19. These were under the care of the same surgeons

Table IV.—Cases of Acute Obstruction in which B. We

No. in Series	Age in Years	Cause of Obstruction	Duration before Operation	Operation	Wh
21	71	Strangulated umbilical hernia ..	72 hours	Division of bands, drainage of sac	
27	51	Volvulus of cæcum	38 hours	Reduction, cæcostomy	
36	15	Bands following appendicitis ..	20 hours	Division of bands	
41	70	Carcinoma of rectum	120 hours	Colostomy	
45	29	Volvulus of colon	56 hours	Colectomy, anastomosis, sigmoid to ascending colon	
51	60	Carcinoma of sigmoid colon	72 hours	Colostomy	
52	56	Band	52 hours	Division of band	
53	62	Strangulated femoral hernia	40 hours	Radical cure	
56	46	Strangulated inguinal hernia	12 hours	Radical cure	
57	23	Volvulus of small intestine	63 hours	Resection 2-ft. ileum, ileosigmoidostomy	
59	43	Strangulated internal hernia	72 hours	Reduction	
65	14	Bands following acute appendicitis ..	120 hours	Ileo-transverse anastomosis	24 hou
66	38	Carcinoma of rectum	36 hours	Sigmoid colostomy	
67	69	Strangulated femoral hernia	48 hours	Radical cure	
68	58	Internal strangulation	168 hours	Reduction	
70	42	Bands	96 hours	Division of bands, ileostomy ..	
72	66	Strangulated femoral hernia	36 hours	Radical cure	
73	55	Strangulated femoral hernia	120 hours	Resection of small intestine, radical cure	24 hou
76	52	Carcinoma of splenic flexure	96 hours	Transverse colostomy	

who took part in the therapeutic test of the use of *B. welchii* antitoxin in cases of acute appendicitis. The cases were unselected and consecutive, with the exception of two which are not included. These two, one of strangulated femoral hernia, one of internal strangulation in an intestinal hernia, appeared before operation to be perfectly fit, but, during the concluding stages of the operation for relief, became suddenly collapsed and died before serum could be administered. Both were healthy men, 53 and 51 years of age respectively.

The mortality of acute obstruction, like that of acute appendicitis, depends largely upon local conditions. It is, moreover, very difficult to eliminate

B. WELCHII ANTITOXIN WAS ADMINISTERED.

WHEN ANTITOXIN FIRST ADMINISTERED	RESULT	REMARKS
At operation	Recovery	Abscess in sac and perforation of gut
At operation	Recovery	Severe toxæmia
At operation	Recovery	Severe toxæmia
At operation	Recovery	Great distention
At operation	Recovery	Obstruction only, no strangulation
At operation	Recovery	Great distention and severe toxæmia
At operation	Death 6th day after operation	Streptococcal septicæmia (*see note, p.* 314)
At operation	Recovery	Severe toxæmia in feeble subject
At operation	Recovery	——
At operation	Death 5 hours after operation	Extreme toxæmia (*see note, p.* 314)
At operation	Recovery	Considerable small-bowel distention
24 hours after operation	Recovery	Progressive distention, not relieved by pituitary injections or enemata
At operation	Recovery	Severe toxæmia and distention
At operation	Recovery	Feeble subject
At operation	Death 24 hours after operation	Extreme toxæmia (*see note, p.* 314)
At operation	Recovery	Severe distention and toxæmia
At operation	Recovery	Moderate distention
24 hours after operation	Death 6 days after operation	Post-mortem: gangrene of gut (*see note, p.* 315)
At operation	Death 20 hours after operation	Severe distention and toxæmia in bad subject (*see note, p.* 315)

errors due to individual differences of opinion as to what constitutes intestinal obstruction in cases of external hernia, and this fact is important because of the large proportion of such cases. For comparison I have therefore taken figures from the paper to be published by Mr. C. M. Page[20] on the mortality at St. Thomas's Hospital for the five years 1919–23. In this series, and in my series of cases which were used for the therapeutic test of *B. welchii* antitoxin, the classification was done by Mr. C. V. Patrick. In *Table III* the two series are given side by side for comparison. Cases of obstruction due to malignant disease, though arising primarily in the large bowel, are included in the series only when there were acute symptoms, for the reasons suggested on p. 297.

Cases of idiopathic intussusception in infants are excluded, because in the vast majority manifestations of intestinal obstruction do not develop. Mesenteric thrombosis is also excluded, as the cause cannot be removed by operation.

The number of cases in the test series is very small, but it is given for what it is worth. With this reservation the mortality shows a striking improvement.

Details of the 19 cases to which anti-gas-gangrene serum was administered are given in *Table IV*. With the exception of the use of the serum the usual surgical treatment was carried out.

The clinical effects of the administration of serum in cases of acute obstruction were similar to those seen in cases of peritonitis. These clinical signs of improvement may, and commonly do, follow the simple surgical relief of the obstruction; it is consequently only by considering the series as a whole that the clinical effects can be assessed. The following are notes of the five fatal cases.

Details of Fatal Cases.—

Case No. 52.—A man, age 56, with old Pott's disease of the spine and great deformity. He had had an inguinal hernia for many years, and was admitted with a fifty-two hours' history of pain and vomiting coming after replacing the hernia. He was a poor surgical risk, and appeared very ill. Forty c.c. of *B. welchii* antitoxin and a pint of normal saline were given intravenously before operation. Laparotomy was performed, and the bowel found caught over a band at the internal orifice of the inguinal hernia. The gut was viable, and was returned to the abdomen. Twenty-four hours after operation the patient appeared to be in excellent condition, with the exception of some post-anæsthetic bronchitis. The bowels were opened twice naturally, and there was no distention. Forty-eight hours after operation he developed a streptococcal cellulitis at the site of the hypodermic injection into the arm, and died four days later. A pure culture of hæmolytic streptococcus was grown from the blood before death.

POST-MORTEM EXAMINATION.—The bowel was not distended; death was due to streptococcal septicæmia; there was no hæmolytic staining of the heart or great vessels.

Case No. 57.—A girl, age 23, with sixty-three hours' acute abdominal pain. On admission she appeared to be extremely ill, and an attempt was made to improve her condition with saline infusion and morphia for six hours before operation. Laparotomy was then performed, and the obstruction was found to be due to volvulus of the lower ileum over a band. A portion of gut about 2 ft. long was not viable, and was resected, an end-to-end anastomosis being performed. The distended small intestine above was also anastomosed to the transverse colon. The operation was difficult on account of intestinal distention. Intravenous saline and 60 c.c. of *B. welchii* antitoxin were administered during the operation. The patient was practically pulseless on leaving the table, and died a few hours afterwards without regaining consciousness. Post-mortem was refused.

Case No. 68.—A man, age 58, with seven days' history of acute abdominal pain and vomiting. On admission his condition was very bad indeed. At laparotomy the lower ileum was found strangulated by a band in the pelvis. The gut was very 'sodden', and perforated accidentally. The band was divided, ileo-transverse anastomosis was performed, and the abdomen closed. Sixty c.c. of *B. welchii* antitoxin were given intramuscularly on the table, and a further 60 c.c. sixteen hours later. The patient became delirious twenty-four hours after operation, and died. Abdominal distention was very marked.

POST-MORTEM EXAMINATION.—There was little peritonitis. The small intestine was grossly distended. The heart and aorta showed an intense hæmolytic staining.

Case No. 73.—A man, age 55, with a five days' history of strangulation of a femoral hernia. At operation a loop of small gut was found to be gangrenous, and 4 in. were resected. After operation the patient did not improve, constipation was absolute, and distention became more marked. During the succeeding four days six injections of pituitary and repeated enemata failed to bring about any relief. *B. welchii* antitoxin was administered intramuscularly in doses of 40 c.c. daily, from the second day until the patient died on the sixth day after operation. No action of the bowels was obtained, and distention did not appreciably diminish. There was a general icteric tinge during the later stages.

POST-MORTEM EXAMINATION.—There was great distention of the small intestine, and about six inches of gut above the site of resection were gangrenous. There was some peritonitis in this area. The heart and great vessels showed slight hæmolytic staining.

Case No. 76.—A man, age 52, who had suffered from 'asthma, bronchitis, and a weak heart' for years. He had had chronic obstruction for 'some weeks', becoming acute for four days. On admission his condition was bad and distention very marked. At operation a carcinoma of the splenic flexure was found, and a transverse colostomy was performed. Sixty c.c. of *B. welchii* antitoxin were given on the table. He did not regain consciousness, and died twenty hours later. A post-mortem examination was refused.

The fatal issue in *Cases* Nos. 52 and 73 cannot be regarded as evidence of failure of the antitoxin. Such cases, however, occur in any series and contribute to the general mortality.

CONCLUSIONS AS TO THE THERAPEUTIC VALUE OF B. WELCHII ANTITOXIN IN CASES OF INTESTINAL OBSTRUCTION AND PERITONITIS.

1. In cases of peritonitis with paralytic obstruction there was a marked clinical improvement as the result of administration of *B. welchii* antitoxin. In cases of organic obstruction similar effects were obtained, but in individual cases it was, as a rule, impossible to differentiate between the effects of the serum and the effect of successful operative relief of obstruction.

2. There is a great improvement in the mortality-rate in the test series of cases to which antitoxin was administered when necessary. In 256 cases of acute appendicitis the mortality was 1·17 per cent as opposed to the normal mortality of 6·3 per cent; in 54 cases of acute intestinal obstruction it was 9·3 per cent as opposed to the normal rate of 24·8 per cent.

3. The beneficial effect of *B. welchii* antitoxin is evidence of absorption of *B. welchii* toxin in cases of intestinal obstruction, whether organic or paralytic.

DOSAGE AND INDICATIONS FOR TREATMENT WITH B. WELCHII ANTITOXIN.

The therapeutic use of *B. welchii* antitoxin is in the experimental stage. The following notes are included for the use of those who wish to test its value.

It is not suggested that the use of this antitoxin is in any way alternative to the recognized primary surgical treatment of intestinal obstruction and of peritonitis by the relief of obstruction and the removal of the cause of peritonitis. It is advocated as a means of combating toxæmia of intestinal origin consequent upon stagnation of the contents of the small intestine, a toxæmia which cannot otherwise be dealt with successfully.

The administration of horse-serum antitoxin has, however, certain disadvantages: (1) It renders the patient sensitive, so that when serum may be necessary afterwards for some other condition, its administration may be more difficult. (2) It may produce rashes and other phenomena which are distressing, though not dangerous. (3) There is the remote risk of anaphylaxis. (4) It is expensive, and, to be of use, large amounts must be given.

The vast majority of cases of acute appendicitis will recover by treatment on the ordinary surgical lines. In cases which develop signs of intestinal obstruction, however, the mortality is very high, and the disadvantages of serum therapy are, by comparison, negligible. In cases of acute intestinal obstruction of the forms discussed, the mortality is normally high—between 20 and 30 per cent—and I suggest that serum should be used as a routine whenever there is evidence of small-bowel involvement. Further, it is not uncommon to see cases which appear to be quite fit before operation, but become rapidly worse after the relief of the obstruction, like the two cases already mentioned, so that it is impossible to predict how any given case will behave. This sudden and unexpected turn for the worse may be due to the liberation of the highly toxic contents of the obstructed intestine and the rapid absorption of a lethal dose of toxin. The only way to combat this eventuality would be the intravenous administration of an adequate dose of antitoxin before operation, if necessary intravenously, on the table.

The effect of antitoxic serum is to neutralize the toxin after its absorption from the intestine. Absorption of the toxin will continue until the small intestine is emptying normally. It is important therefore to recognize, particularly in paralytic obstruction due to peritonitis, that a clinical improvement is not an indication for stopping the administration of serum, which must be continued until the small bowel is emptying itself regularly and effectively.

In the therapeutic use of antisera it is essential that the dose should be adequate. The disadvantages of the administration of horse-serum are not increased with the size of the dose, except as regards cost. There is no maximum dose, but there are minimum doses, the administration of which is futile. The younger the patient the higher the mortality of peritonitis, and so the greater the necessity for an adequate dose of antitoxin. Further, anaerobic toxin can produce severe cardiac degeneration, and it is essential that the antitoxin should be administered before this has gone too far to be recoverable.

In cases of peritonitis patients are gravely ill, in part, according to the hypothesis, on account of the intestinal toxæmia, but also on account of their peritonitis. For the direct treatment of the latter, the use of anti-gas-gangrene serum contributes nothing, and inevitably cases must occur which die on account of the peritoneal suppuration, apart from intestinal toxæmia, though no such cases occurred in this series. It is well known, however, that the peritoneum has extraordinary powers of dealing with infection, and, if the intestinal toxæmia can be kept in check by the administration of *B. welchii* antitoxin, in the vast majority of cases, if given time, the patients will overcome their peritonitis.

The serum used was anti-gas-gangrene serum containing *B. welchii* and *Vibrion septique* antitoxins, series G 155, prepared by Messrs. Burroughs, Wellcome & Co. for therapeutic use. The serum is of higher titre than the

gas-gangrene serum used during the war, and has been found to be of value in treating cases of gas-gangrene resulting from street accidents. It is hoped that a more concentrated serum will shortly be available.

In consideration of the above, it is suggested that, in cases of peritonitis, *B. welchii* antitoxin should be administered to those patients who show evidence of intestinal paralysis and anaerobic toxæmia, as manifested either by severe or progressive abdominal distention, increasing pulse-rate, restlessness, or repeated vomiting, especially if offensive or brown, particularly if these signs appear after surgical treatment has been carried out. In extreme cases the administration of serum should be begun at the earliest possible moment—if practicable, before operation. The initial dose of serum to be given should be at least 80 c.c. intramuscularly, and in extreme cases an additional 40 c.c. intravenously. After the initial dose 40 to 80 c.c. should be given intramuscularly every day until the bowels are moving spontaneously and regularly and distention has almost or completely gone, and absorption of toxin consequently been brought to an end. The patient should be kept 'desensitized' against horse-serum until all risk is passed of the development of obstruction, which is apt to come on as the result of peritoneal matting during convalescence.

In cases of acute intestinal obstruction indications for the use of antitoxin are, broadly speaking, the same as those given in the previous paragraph in connection with cases of peritonitis, though the abdominal distention may be caused sometimes by the large bowel alone. The clinical appearances, however, of cases of acute intestinal obstruction are apt to be deceptive, in that, as already mentioned, cases showing no evidence of toxæmia before operation not infrequently die rapidly with toxæmic symptoms soon after successful operative relief. Therefore I would suggest that the only way of avoiding unexpected failures is by intravenous administration of 40 c.c. of antitoxin prophylactically before operation, preferably with saline solution if there is much dehydration, and an additional 40 c.c. of serum intramuscularly. Afterwards, as in cases of peritonitis, the administration of serum should be continued until normal evacuation of the bowel is re-established. I have myself at times used smaller doses than those here advocated, but the series was experimental, and patients were kept under constant personal observation. Latterly I have been convinced that the use of larger doses is the only way of securing full benefit of the serum.

RESULTS OF PREVIOUS INVESTIGATIONS OF THE TOXÆMIAS OF ACUTE INTESTINAL OBSTRUCTION.

A vast amount of research has been done on the toxæmia of acute intestinal obstruction, and the literature on the subject is correspondingly extensive. I will refer only to such work as I have been able to find which appears to bear on the aspect of the subject under discussion.

Murphy and Brooks,[21] Dragstedt,[22] and others have shown that the presence of bacteria in the obstructed intestine is necessary for the development of toxæmia. Stone and Bernheim[12] and Dragstedt[23] were able to obtain from the contents of obstructed intestine in dogs material which was toxic to

animals on intravenous injection. This toxic substance was extremely stable, withstood heating to 70° C. for one hour, and was not destroyed by filtration. Dragstedt was unable to demonstrate the development of any immunity in animals which had been subjected to repeated sublethal doses, and suggested that the poison was a product of bacterial fermentation of the histamine type and not a true bacterial toxin. No mention was made of the reaction of the material injected, which must be an important factor when relatively large amounts are introduced intravenously.

Using Berkefeld filtration for the sterilizing of intestinal contents and intramuscular injections, I have never found any evidence of the toxin described by these workers. It therefore seemed possible that the method of sterilizing the material for animal inoculation by heating to 70° C. might be responsible for the liberation of the toxic substance. With a view to testing this possibility the following experiments were carried out.

Intestinal contents obtained by jejunostomy from two human cases, one of obstruction due to bands, one of peritonitis, were divided into two portions. One portion was prepared as already described for examination for the presence of *B. welchii* toxin, by Berkefeld filtration in the cold; the other was heated at 70° C. for one hour before filtration, as described by the American workers. Each portion was then inoculated into two groups of mice, one group receiving in addition *B. welchii* antitoxin, the other group normal horse-serum, as in the tests for the presence of *B. welchii* toxin. The results are represented together in *Table V*.

Table V.—Results of Tests for B. Welchii Toxin in Intestinal Contents in Mice.

Source of Material	Cold Filtrate of Intestinal Contents				Heated Filtrate of Intestinal Contents			
	Mice injected with filtrate and *B. welchii* antitoxin		Mice injected with filtrate and normal horse-serum		Mice injected with filtrate and *B. welchii* antitoxin		Mice injected with filtrate and normal horse-serum	
	Total	Died	Total	Died	Total	Died	Total	Died
Obstruction by bands	6	0	12	2	6	3	12	5
Peritonitis	5	0	10	5	6	3	8	4
Totals	11	0	22	7	12	6	20	9

It appears, therefore, that in these two cases a toxic substance was liberated from the intestinal contents by heating. This substance was not neutralized by *B. welchii* antitoxin, and was not present in the unheated material. Further investigation is being made on this point.

It is interesting to find that in 1904 Kamen is reported by Herter[24] as having obtained *in vitro* soluble thermostabile poisons from *B. welchii* cultures. The effects of these poisons when inoculated into rabbits closely resembled the effects obtained on animal inoculation of the 'toxin' obtained from obstructed intestine by Stone, Bernheim, and Dragstedt. These poisons could not, of course, be the true exotoxin of *B. welchii*, which is rapidly destroyed by heat, and could not have survived the method of preparation employed by these workers.

DISCUSSION.

The investigation which is described in this paper was begun on the hypothesis that the toxæmia of acute intestinal obstruction, whether organic or paralytic, is, in part at least, due to the absorption of anaerobic toxins from the stagnant contents of the small intestine. It is not asserted that *B. welchii* is the only toxin, but it was the only one of which evidence was found during this investigation. It has not been possible to produce absolute proof of the hypothesis as a whole, but in each particular line of approach evidence has been found in support of the probable truth of the original assumption. Failures have been readily explicable by the limitations imposed by the nature of the materials under investigation and the difficulties of technique. Further, the hypothesis is in complete accordance with the known clinical features, and, if it is true, offers a simple solution for many hitherto unexplained problems.

The remarkable difference between patients suffering from absolute constipation even for weeks, with nothing more untoward than lassitude, a furred tongue, and a headache, and a patient with acute obstruction of the intestine, is explained by the fact that in the former there is no material stagnation above the ileocæcal valve, and hence there is no formation of *B. welchii* toxin. Similarly, the complete absence of toxæmia in cases of pyloric obstruction, and the almost invariable and immediate benefit which results from operative relief, are explained by the fact that in the stomach the aerobic acid conditions prevent the formation of *B. welchii* toxin. Even in pyloric obstruction due to malignant disease, when as a rule hydrochloric acid is absent, the reaction is still usually acid. It is possible, in those rare cases in which the reaction becomes neutral, that the proliferation of *B. welchii* and the development of toxæmia might occur. A case of 'acute hæmorrhagic gastritis' which came under observation recently confirms this possibility.

The relatively high mortality of acute obstruction of the small intestine by bands and adhesions—a mortality which has not altered materially for many years—coincides with the presence of ideal conditions in the small intestine for the proliferation of *B. welchii* and the formation of this toxin. Further, the sudden unexpected deaths after most successful operative relief of small-bowel strangulation may be explicable on the hypothesis by the rapid absorption of a lethal dose of toxin from the contents of the obstructed loops after their release.

The most difficult feature of intestinal obstruction to overcome successfully is paralysis of the small intestine. In cases of organic obstruction paralysis persists often after removal of the organic cause. In peritonitis the obstruction may be entirely paralytic, though at times, in part at least, it may be organic. Many procedures have been advocated with a view to overcoming this paralysis, and emptying the obstructed bowel. Injections of pituitary and eserine, and drainage of the small bowel by ileostomy, jejunostomy, or ileocolostomy, have all been tried with varying success; and success or failure in any case probably depends upon whether the bowel is still able to contract or not. If the bowel has lost the power of contraction, pituitary is ineffective, and drainage will only empty the loop into which an opening

is made, and the results of drainage are frequently so disappointing in this respect that these methods have not received general acceptance. Of five cases in which ileostomy was performed for peritonitis or obstruction, sufficient intestinal contents for investigation for the presence of *B. welchii* toxin was obtained only twice, and to these two cases *B. welchii* antitoxin was being administered in large amounts. Now the toxin of *B. welchii* has a powerful effect upon muscle tissue, and this paralysis of the intestine may well be due in part to the presence of *B. welchii* toxin in the contents, to which the intestinal wall is exposed in its greatest concentration. The administration of antitoxin, besides combating the general toxæmia, may help to overcome the small-bowel paralysis. In the absence of organic obstruction, if the intestine is able to contract, absorption will cease on the evacuation of the toxic contents to the exterior, or into the large bowel, where the toxin is destroyed by the acid reaction.

It was observed repeatedly, during the clinical trial, that, after the administration of *B. welchii* antitoxin, distention rapidly diminished or disappeared, and the bowels were opened spontaneously. As these effects always coincided with clinical improvement, it is impossible to attribute them directly to the antitoxin with certainty, and further investigations are therefore being carried out.

The striking difference between the gravity of the clinical condition of the patient suffering from the presence of pus in the peritoneal cavity and that of a patient suffering from the presence of pus in another situation, such as the pleura, has long been the subject of speculation. The same difference is found when there is a localized collection of pus in the abdomen, where the area of contact with the small intestine is small, in contrast to when there is a diffuse peritoneal infection, where the area of contact with the small intestine is great. Peculiarities of absorption from the peritoneum have been postulated and rejected. More recently the importance of the intestinal stagnation which is associated with peritonitis has been emphasized by many writers, but the nature of the resultant toxæmia has not been investigated. This hypothesis offers some explanation for these differences, in that the degree of stagnation and consequent production of *B. welchii* toxin depends upon the extent of small intestine affected by inflammation.

The literature of intestinal intoxication is immense, but, so far as I am aware, experimental results have all been negative, with the exception of the work of the American investigators already discussed. I believe that the experiments described in this paper are the first to indicate the presence of a true toxin of intestinal origin, its nature, and the conditions necessary for its production. The hypothesis, if correct, opens a wide field for investigation. Therapeutically, if subsequent results are as encouraging as those reported in this paper, the application of the hypothesis is important, because of the large number and variety of cases in which stagnation may occur in the small intestine as the result of organic or paralytic obstruction. Such cases have at present a high mortality, which may be materially reduced by the use of antitoxin.

The only possible objection to the hypothesis that I have been able to find is the rarity in cases of obstruction or peritonitis of the occurrence of

generalized *B. welchii* infections, giving rise to metastatic lesions and the finding of 'foaming liver' post mortem. 'Foaming liver' is a phenomenon resulting from post-mortem changes in a body in which there has been a general dissemination of *B. welchii* before death. It is not a very frequent occurrence in laboratory experiments, and was not common in human cases of gas-gangrene infection during the late war. I have, however, seen 'foaming liver' in five human cases of puerperal infection with *B. welchii*, and in one case of typhoid fever. I have never seen a 'foaming liver' from a case of obstruction or peritonitis, and have only once seen a metastatic lesion arising in the 'pressure points' of the back in the case of appendicular peritonitis. Local post-operative infections of the abdominal wall with *B. welchii* are not so uncommon, but are by no means frequent. These local infections must result from direct fæcal contamination of the wound, or contamination with fæcal pus, which very frequently contains *B. welchii*; either of these must be a very common occurrence. I can only suggest that it requires very particular conditions for the establishment of *B. welchii* infection in living human tissues, the most important probably being an impairment of the blood-supply; and the rarity of the establishment of *B. welchii* infections of the abdominal wall lends support to this view. Further, the organism is almost ubiquitous, in dust, in soil, and milk; yet in civil practice gas-gangrene is extremely rare, even after street accidents, unless there has been great tissue damage. Growth in a hollow viscus, such as the intestine, outside the sphere of the influence of the living tissues, is an entirely different matter, which under suitable conditions has been shown in this paper to occur readily, and absorption of the soluble toxins may easily take place without any invasions of the tissues by the bacillus.

SUMMARY.

1. The hypothesis put forward in the paper is that the toxæmia in cases of intestinal obstruction, whether organic or secondary to peritonitis, results, in part at least, from the absorption of the toxin of *B. welchii*, due to the proliferation of this anaerobe in the stagnant contents of the small intestine.

2. The clinical resemblance of the toxæmia of intestinal obstruction and peritonitis to that of gas-gangrene is discussed.

3. Evidence is given of the proliferation of *B. welchii* in the small intestine in cases of intestinal obstruction and peritonitis in human subjects and in these diseases experimentally produced in dogs.

4. Evidence is given of the presence of *B. welchii* toxin in the small intestine in intestinal obstruction and peritonitis in human subjects and in these diseases experimentally produced in dogs. The toxin was not found in normal small intestine contents, or in contents of the large intestine even when obstructed, in human subjects.

5. Evidence is given compatible with the absorption of *B. welchii* toxin, from the clinical aspect of cases, from the occurrence of hæmolysis, and from microscopical changes in the heart and liver of fatal cases.

6. A therapeutic test of the hypothesis by the administration of *B. welchii* antitoxin in a series of cases of appendicular peritonitis and acute intestinal obstruction is reported.

7. Evidence is given, in the results of the therapeutic test, of clinical benefit to individual cases, and of improvement of the general mortality-rate in each series.

8. Practical details are given of the indications for the use of *B. welchii* antitoxin, and of dosage for the therapeutic test of the hypothesis.

9. Previous investigations of the toxæmia of acute intestinal obstruction are discussed, and a possible reason for their disagreement with the results of this investigation is suggested.

10. The results of the investigation and the theoretical and clinical applications are discussed.

Finally, I have to thank those whose assistance enabled this investigation to be carried out. As I knew little of bacteriology and nothing of anaerobe technique, the amount of help which I required was very great. In addition to those mentioned in the text, I am especially indebted to the following :—

Dr. S. C. Dyke, late Pathologist to the Surgical Unit, but now Pathologist to the Wolverhampton General Hospital, with whom the work was begun ; Miss Muriel Robertson, of the Lister Institute, and especially Dr. R. A. O'Brien, of the Wellcome Research Institute, for his generous assistance with sera and *B. welchii* toxins ; Sir Cuthbert Wallace, Director, and Mr. B. C. Maybury, Deputy Director of the Surgical Unit, St. Thomas's Hospital, who have given constant advice, suggestions, and criticism ; Professor L. S. Dudgeon, Director of Pathology at St. Thomas's Hospital, for advice and criticism during the concluding stages of the investigation.

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A BONY TUMOUR OF THE BREAST.

By S. C. DYKE, Wolverhampton.*

Osteoid tumours are among the rarest of those occurring in the breast of the human being. So rare are they that although they were mentioned by Cooper, Nélaton, Cruveilhier, and Leser, their very existence was doubted by Billroth. The literature now, however, contains reference to a fair number of them. Deaver and McFarland[1] (1918) in their work, *The Breast*, take cognizance of these tumours and give a full bibliography. Lange[2] (1881) describes an 'osteo-chondroma' of the female breast; when removed this tumour was the size of a nutmeg, and had taken thirteen years to increase to this size from that of a berry. Happel[3] (1895) describes a chondroma of the breast which attained the size of a goose egg a year from the time at which it was first noticed; thereafter it remained stationary for nine years, when it started to grow again and rapidly attained the size of a baby's head. Morton[4] (1904) describes a small tumour removed, together with the breast and glands, which on examination proved to be a " spindle-celled sarcoma with areas of cartilage some of which was calcified." This is one of the few cases in which there is any later history; fifteen months from the time of operation a mass of similar growth was removed from the buttock, and later a like growth appeared on one finger. Morton states that five similar cases had been shown at the Pathological Society of London within the previous five years. Cornil and Petit[5] (1905) describe a large nodular tumour removed from the mamma of a bitch; it consisted of areas of cartilage and bone formation scattered throughout the mammary tissue, and was considered by the authors to be due to a diffuse chronic inflammatory condition. They state that the condition is not uncommon in bitches. Sick[6] (1906) describes a fatty tumour removed from a breast which contained numerous small points of cartilage formation, each surrounded by a fibrous capsule. Chevrier and Delval[7] (1910) describe a 'fibro-myxo-chondro-ostéome' the size of a walnut removed from the female breast; it consisted of three tumours in coalescence, and was delimited, mobile, and non-adherent. Sehrt[8] (1907) describes a tumour consisting of miscellaneous and giant cells rich in chromatin, cartilage, calcification, and in certain areas showing bone formation. McIver[9] (1923) describes a mass the size of a walnut removed from the breast of a woman aged 60. It consisted of " nondescript connective tissue and showed numerous mitotic figures. There were trabeculæ of osteoid tissue scattered throughout it and occasional areas of cartilage. In one area an alveolar process with teeth could be made out ". Kreibig[10] (1925) describes a tumour the size of a walnut the matrix of which consisted of spindle and giant cells and in which cartilage and bone

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were present; in one place was an area of 'carcinoma simplex'. He regarded the tumour as an osteosarcoma in combination with a carcinoma. Fowler[11] (1925) reports an osteochondroma of the breast occurring in a woman aged 67. Apart from the presence of cartilage and bone no mention is made of the cellular nature of this tumour. It had been present for ten months, and when excised proved to be 3 by 5 cm. in size.

PARTICULARS OF THE CASE.

Clinical History.—The patient from whom the tumour to be described was removed, was a single woman, age 50. She first noticed a lump in the left breast in December, 1924, when it appeared to be about the size of a hazel-nut; it remained about the same size and gave no inconvenience up to May, 1925, when it commenced to grow, the rate of growth becoming more and more rapid up to August of the same year, when the increase in size was, according to the patient, daily perceptible.

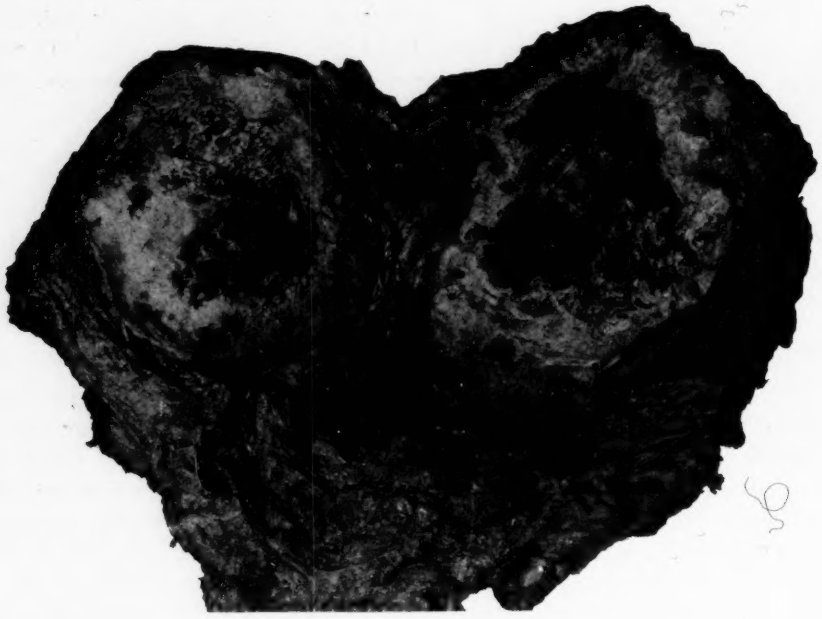

FIG. 187.—Photograph of tumour.

The breast was removed by Mr. S. Maslen Jones, F.R.C.S., at the Hospital for Women, Wolverhampton, in September, 1925, the whole organ being then about the size of a baby's head. Little doubt was entertained at the time of the operation but that the tumour was a carcinoma, and the axillary glands were cleared. The patient made a good recovery.

Description of Specimen.—The specimen as received in the laboratory consists of a greatly enlarged breast. Lying immediately under the nipple is a tumour, roughly circular and about 10 cm. in diameter. This tumour lies below upon the surface of the great pectoral muscle, which it does not invade, and extends above to the subcutaneous fat. Between this fat

and the tumour there exists a rather indefinite line of cleavage; this is not difficult to distinguish where the tumour comes into contact with the subcutaneous fat, but disappears where it lies over the muscle. In the centre of the tumour is a large cavity (*Fig.* 187) with ragged edges, roughly oval and having diameters of 5·0 and 2·5 cm. At operation this cavity was filled with blood-clot and débris, but had been evacuated when the specimen was received at the laboratory. The ragged nature of the edges of the cavity is due to the presence of numerous spicules of bone, and the whole tumour is firm in consistency and cuts in such a manner as to suggest the presence of bone. The outer surface of the tumour presents a greyish-white appearance. On making section through the whole breast, in addition to the large mass with a cavity in the centre, several smaller masses of the same tissue, showing extravasation of blood into their centre but no cavity, are apparent lying in the fat surrounding the central tumour. On superficial observation it appears as though one were dealing either with a tumour consisting of both fat and ossifying tissue, or of one large and several smaller ossifying tumours scattered throughout the fatty substance of the breast. On more careful examination it becomes apparent that the actual state of affairs consists of one large ossifying tumour, smooth at one of its edges, but with a number of irregular protrusions on the other side, these forcing their way into the fatty tissue of the breast, and giving on cross-section the appearance of separate tumours unconnected with the main mass.

FIG. 188.—Periphery of the tumour, showing absence of capsule, and cells of spindle and giant type.

On the smooth side of the tumour and under the nipple the edge of the tumour tissue is more or less definitely marked, but the boundaries of the projecting portions of the tumour shade almost imperceptibly into the surrounding fatty tissue.

Microscopical examination of the tumour shows that the most active and vegetative portion is the periphery (*Fig.* 188). Here two types of cell can be distinguished; the most plentiful, and that forming the bulk of the tumour tissue in this area, is a large spindle or round cell with a reticular type of nucleus. Interspersed among these, and increasing in number and size as the actual edge of the tumour is reached, are giant cells containing many small and deeply staining nuclei. The largest of these latter cells occur at the extreme periphery of the tumour where it abuts upon the normal tissue. In appearance they are indistinguishable from the giant cells of 'myeloid sarcoma' or from osteoblasts, or osteoclasts.

A short distance in from the edge of the tumour evidence of bone

formation commences (*Fig.* 189). Areas are present in which calcium salts are being deposited in the intercellular tissue, apparently without previous formation of cartilage.

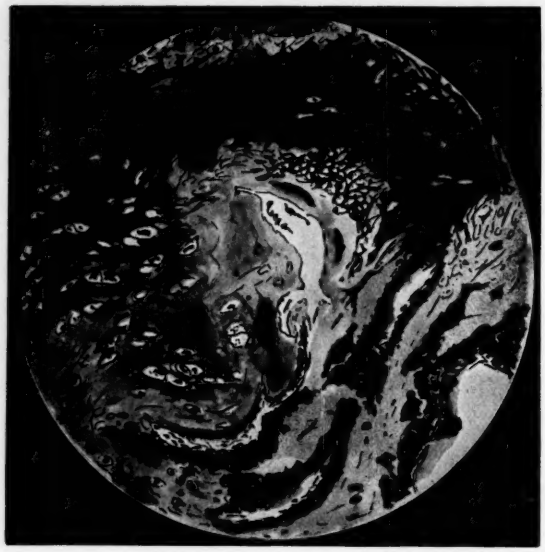

FIG. 189.—Portion of tumour deep to that shown in *Fig.* 188, showing formation of cartilage and bone.

In other places cartilage is being formed and calcified at the same time. None of the sections examined shows any considerable mass of cartilage in which calcification is not actively in progress. In no case is the process of calcification seen to be occurring in the immediate neighbourhood of the giant cells, and it would appear that it is not they but the spindle cells which are actively concerned in this process.

As the central portion of the tumour is approached, the consistency becomes harder. Decalcification was necessary before microscopic sections could be cut of the tissue abutting upon the central cavity. This tissue shows ample evidence of ossification (*Fig.* 190). The general arrangement is loosely cancellous; many of the bony trabeculæ show lacunæ, some of which contain the remains of cells which may presumably be regarded as bone corpuscles. The trabeculæ present no proper laminar structure; the spaces between them are filled in with necrotic cell débris; there is no attempt at marrow formation, and the remains of such cells as are distinguishable suggest that the original inhabitants of the intertrabecular spaces were of the spindle type described as constituting the mass of the tumour at the periphery. The tumour is obviously dying at the centre and increasing in size by growth at the periphery.

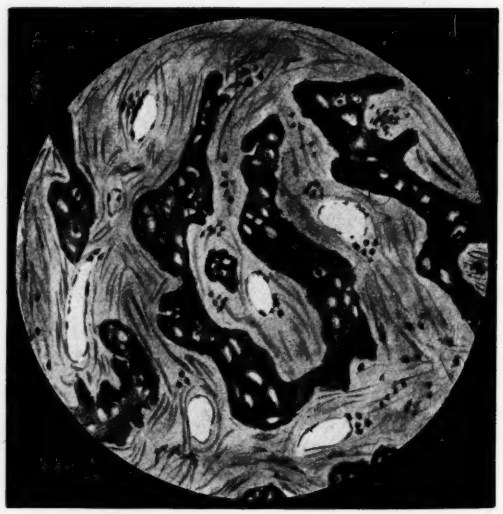

FIG. 190.—Portion of tumour abutting upon central cavity, showing well-formed bone and absence of cellular elements.

Microscopical examination of the tumour indicates that increase in size is taking place by a process of pushing back of the surrounding tissue rather than by definite invasion. The surrounding tissues show a considerable condensation of fibrous tissue,

almost sufficient in places to merit the name of a capsule. In other places this capsule formation is not so well marked, and spread of the tumour is taking place by invasion of the surrounding tissues by the cells of the tumour. Even where the fibrous-tissue capsule is fairly well marked, as in the portion illustrated, spread of the tumour cells into this fibrous tissue is quite evident. The appearances suggest a mild degree of malignancy.

Portions of the breast tissue abutting upon the tumour show marked change of the type usually described under the name of 'chronic mastitis'. These changes comprise dilatation of the acini, proliferation of their epithelium, and in some places the actual formation of new acini. In some areas cyst formation is a marked feature. There is nothing to suggest malignancy in the portions cut, though by some authorities the changes would perhaps be described as precancerous.

An enlarged gland removed from the axilla showed evidence of chronic inflammation, but none of malignancy.

Up to the time of writing, nine months from the time of the operation, there has been no recurrence of the tumour.

DISCUSSION.

The appearance in the breast of a tumour composed of such foreign structures as cartilage and bone seems difficult of explanation. Leaving aside the occurrence of metastases from tumours elsewhere, which would appear to be out of the question in any of the cases mentioned above, two explanations may be offered.

1. The condition may be a metaplasia following upon a chronic inflammation rather after the fashion of myositis ossificans in chronically irritated muscle.

2. The presence of the foreign cells may be the result of proliferation of totipotential cells which had become included early in embryonic life. In other words the tumours may be teratomata.

The first explanation may apply to certain of these cartilage- and bone-containing tumours. It is the view adopted by Chevril and Petit of the nature of their tumour in the bitch, and the description of Sick's tumour would suggest that his was something of the same sort. It is difficult to see how it can apply to many of the other similar tumours described above and to the tumour forming the subject of this note.

The second explanation would seem to fit in best with the case here described, and with most of those recorded in the literature. It is suggested by Ewing that the majority of these tumours are teratomata and fall into the same category as the mixed tumours of Wilms. The specimen described by McIver containing teeth lends very strong support to this view of the case. It is at first sight rather difficult to accept a tumour apparently consisting entirely of cartilaginous and osseous tissue as being teratoid in nature. One is in the habit of associating the teratomata with a mixture of many types of cells. It is, however, no unusual occurrence to meet with a teratoid tumour in which one type of cell has proliferated to a far greater extent than any of the others.

This is particularly the case where malignant change supervenes in one group of cells in a teratomatous tumour. Professor Haswell Wilson has pointed out to me that a very excellent example of this, of fairly common occurrence, is the overwhelming growth of cells of the chorionepithelioma type which may occur in teratomatous tumours of the testicle.

Further evidence of the teratomatous nature of these tumours would seem to be offered by Kreibig's case, in which areas of carcinoma were present. Kreibig was careful to point out that the appearances were not those of a sarcomatous change in a carcinoma, as described by Kettle[12] (1919), but a definite inclusion of areas of carcinoma within a sarcomatous tumour. The only circumstances capable of producing this state of affairs would seem to be the occurrence of malignancy in a teratomatous tumour.

It would seem probable that in the tumours of the type described above we are dealing with teratomatous mixed tumours of the breast in which one group of cells has assumed a speed of growth out of all proportion to that of the other elements present. This rapid proliferation must lead to a suspicion of malignancy. Morton's tumour demonstrated its malignancy by producing metastases, but this seems to have been the only case in which such definite proof has been forthcoming. Sehrt regarded his tumour as malignant on histological grounds. In the case described above evidence of a mild degree of malignancy was afforded by the invasion of the surrounding tissue by the tumour cells, though only to a slight extent, and the comparative failure of capsule formation.

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THE EXTRAPERITONEAL CLOSURE OF ARTIFICIAL ANUS (GREIG SMITH'S METHOD).

By T. CARWARDINE, Bristol.

In 1895 the late J. Greig Smith published in the *Bristol Medico-Chirurgical Journal* a method of extraperitoneal closure of artificial anus,[1] and again in his book on abdominal surgery.[2] If certain points of importance in technique were understood, the method would be more popular; consequently a detailed description of the operation may be acceptable, with observations that are the outcome of experience, and a summary of results.

In Greig Smith's words: "The aim of the operation is to perform enterorrhaphy without opening the general peritoneal cavity; and this is managed by detaching from the parietes all round the fistula and anus sufficient peritoneum to permit delivery of the gut through a parietal incision without separating it from its peritoneal adhesions."

PRELIMINARY TREATMENT.

Preliminary treatment is necessary, and consists of division of the spur and restoration of a natural channel by mechanical means.

Division of the Spur.—There is no better or safer device for division of the spur than Dupuytren's enterotome if proper precautions are taken. Before its application the depth of the spur should be estimated by inserting the two gloved forefingers into the anus. Care should be exercised to select an enterotome the blades of which approximate first at a part corresponding with the apex of the spur. After the spur is clamped sufficiently to retain the enterotome in place the further compression should be gradual, the screw being turned about half a turn daily. It takes five or seven days for the spur to be crushed through. There is some risk of the enterotome being pulled out, hence the range of the patient's hands should be limited by a clove-hitch tied round each wrist and attached to the head-rail of the bed, or by some other means. There is risk also of the enterotome being pushed in accidentally; consequently the instrument should always be exposed to view and protected from accidental pressure. For this purpose a large perforated cork bung may be bisected at its diameter after cutting a groove in its circumference. The two halves are applied below the pivot of the enterotome and are tied together by a strong thread placed in the circumferential groove (*Fig.* 191).

Fig. 191. — Enterotome protected by a divided cork.

Insertion of the Banks Tube.—The ingenious method of Mitchell Banks comprises the insertion of a piece of rubber tubing of considerable dimensions introduced on the stretch. It rests comfortably in the large entering gut, dilates the contracted efferent gut, and presses back any encroaching spur. It is anchored by a piece of wire passed through its lateral wall which prevents the tube from being extruded and from being carried down the bowel (*Fig.* 192). It is inserted as soon as the spur is divided, to prevent the divided halves of the spur from joining again. There should be no haste at this stage; the longer the tube is kept in, the more natural will be the fæcal flow, and the smaller will the artificial anus tend to become, thus making for ultimate success.

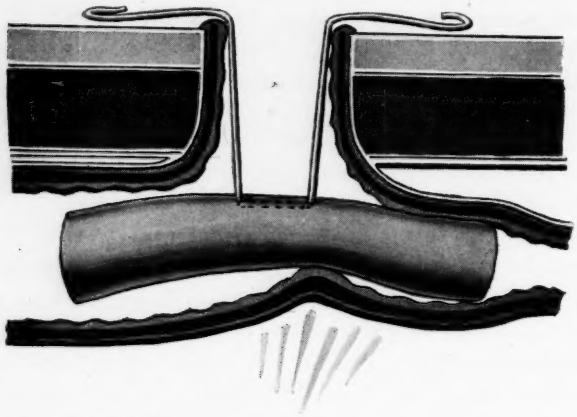


FIG. 192.—The Mitchell Banks tube in position, anchored by silver wire. Sectional view.

Use of the Button-valve.—The natural channel and normal defæcation may be further restored by using a valve to shut off the fæces from the surface. Since 1897 I have employed a valve (*Fig.* 193) of an oval piece of sheet rubber, or portion of a large drainage tube from $\frac{1}{16}$ to $\frac{1}{8}$ in. thick. This is rolled up and inserted into the artificial anus, when it

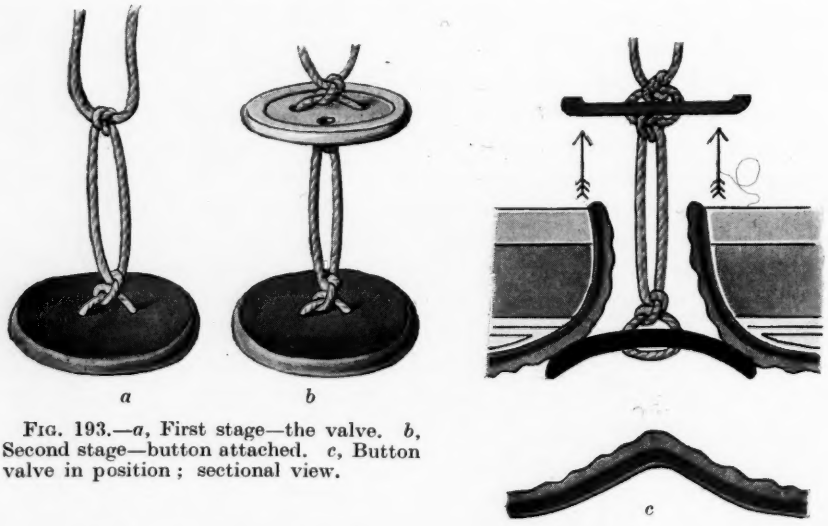


FIG. 193.—*a*, First stage—the valve. *b*, Second stage—button attached. *c*, Button valve in position; sectional view.

opens up within the bowel and acts effectively. Through its centre a double thread or string is passed and knotted, and the string is knotted again at a convenient distance—generally about $\frac{3}{4}$ in. Then a large coat-button is threaded on the double string, which is knotted once again, and

the excess of thread is cut off. When the valve is inserted the button provides a means for supporting it against the internal aperture of the anus, merely requiring some gauze or lint to be wound round the string underneath the button.

The button-valve was used for some of Greig Smith's cases, and it was published and illustrated in 1900. It is a very useful device and may be used elsewhere; by its means the life of a patient with a large gastric fistula has been saved, the fistula closing entirely. A smaller valve may be used as the anus reduces in size. Harrison Cripps employed a roll of lint for a similar purpose, and in 1917 C. N. Dowd described and illustrated an obturator acting on the same principles, the inner valve being made from an ovoid piece of flat wood.[3]

The preliminary mechanical treatment is of very considerable help; indeed, in some cases natural defæcation is restored and the artificial anus becomes very small. It is tedious at first—the only objection to it; but it is safe and generally sure, and extraperitoneal closure can be proceeded with subsequently.

TECHNIQUE OF EXTRAPERITONEAL CLOSURE.

The extraperitoneal cure may be described in four operative stages: (1) *The incision;* (2) *The separation of the bowel;* (3) *The suture of the opening of the bowel;* (4) *The closure of the wound.*

1. **The Incision.**—To quote Greig Smith's words: "The operation is begun, as in that for fistula, by making incisions along the direction of the chief muscular fibres on each side of the opening down to the subperitoneal tissue. The length of the incisions will vary according to the thickness of the parietes, but will not be shorter than two inches on each side of the anus. The knife is carried round the gut adherent to the parietes, liberating it thoroughly." It was Greig Smith's practice to include a certain amount of skin if necessary, which, when inverted, increased the lumen of the bowel at the part; and I have done so in the majority of instances. It matters little in which direction the skin-flaps are taken, but their shape is important in view of subsequent inversion and suture. They are best made of a semilunar shape, the incisions meeting the mucous edge at the two poles of the diameter (*Fig.* 194). When skin is included the incision should not be oval and uniform all round the anus; such a shape makes the suturing and inversion more difficult.

FIG. 194.—The incisions.

2. **The Separation of the Bowel.**—"The peritoneum, with its areolar tissue, is separated from the overlying muscle all round over a circle of two inches radius or more. The bowel, with its adherent parietal peritoneum, is then delivered through the incision. All superfluous pieces of tissue are removed, and the gut is ready for suture." "The most important detail is liberation of the gut by detachment of the parietal peritoneum. If detachment

is well begun at the distal ends of the incisions, and the plane of separation is followed up to the very margin of the fistula or anus, the operation is much simplified." This important liberation of the gut needs some explanation in detail, which we may illustrate in a case of left inguinal anus.

The longitudinal incisions in the skin are deepened until the aponeurosis of the external oblique is recognized with its muscular fibres at the upper angle (*Fig.* 195); its fibres are divided distally and the division is continued to near the artificial anus; then the finger is swept under the external oblique to set it free from the artificial anus and from underlying tissues. Here, and in later stages, Greig Smith's dissecting scissors of proper pattern are very useful. Then the muscle fibres of the internal oblique, incorporated with those of the transversalis, are identified. These are undermined, beginning well to the *distal* ends of the incisions until the subperitoneal tissue is reached, where the forefinger can be inserted and swept round. Near the artificial anus the muscle fibres will be adherent and may be divided there with dissecting scissors or a sharp scalpel; but it is best to insert the finger under the distal part of the muscle and to pass the blades of the scissors between the finger and the edges of the artificial anus

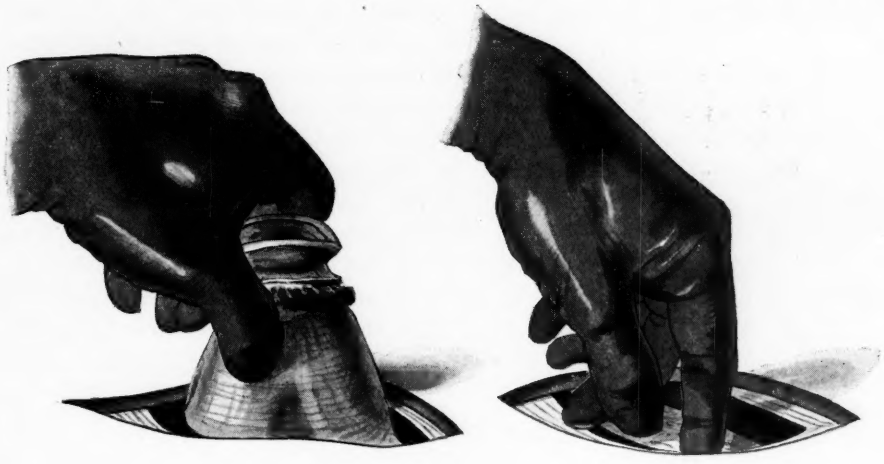
FIG. 195.—Superficial separation of the bowel.

FIG. 196.—Deep separation of the bowel, showing the use of Greig Smith's dissecting scissors and the protection of the peritoneum by the left forefinger.

(*Fig.* 196). If a small hole is made into the peritoneum accidentally, there need be no anxiety, for experience shows that it never leads to trouble. Care should be taken to free the aponeurosis and musculature as separate layers so that they may be approximated later with the least possible tension. It should now be possible—and the test should always be tried—to lift the bowel right out of the wound and to drop it with ease into the

abdomen, although 'extraperitoneal' (*Fig.* 197). If this free mobilization can be carried out, the chances of a successful closure are great. It is the crucial stage of the operation, and the one which has not been appreciated as it should be.

3. **Suture of the Opening of the Bowel.**—All superfluous pieces of tissue are removed. The trimming should be thorough, especially if skin-flaps are



a *b*

FIG. 197.—Free mobilization of the bowel. *a*, The bowel lifted right out of the wound, showing the free mobilization—very important. *b*, The bowel placed within the abdomen, the anus extraperitoneal.

utilized, so that the edges may be inverted without tension. The edges are sutured with inverting continuous catgut stitches, using a round-bodied needle for mucous membrane or a fine bayonet-pointed needle for skin-flaps. It is best to begin the suturing of the opening at each end and to tie the ends of

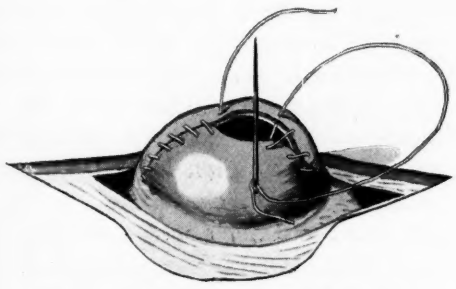


FIG. 198.—Suturing the edges of the opening in the bowel. The two sutures are begun at the ends and tied together in the middle.

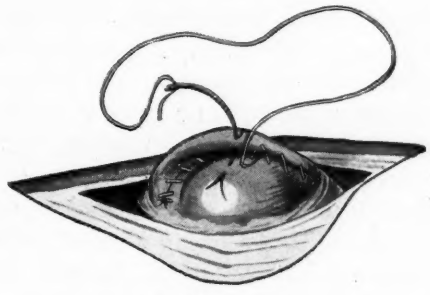


FIG. 199.—Reinforcing suture inverting the tissues and burying the previous suture.

the sutures together in the middle (*Fig.* 198); the intucking is thus more perfect and a terminal pocket which is difficult to invert is avoided. Next a reinforcing continuous catgut suture is passed, Lembert fashion, to invert the edge still further (*Fig.* 199); the tissue is fibrous and a strong hold can

be taken, using a triangular-pointed needle. Then a further reinforcement can be made by a few interrupted sutures. "Particular care is given to the deep suturing. A good hold of the tissues is taken, and each stitch must bring about accurate apposition." A trace of antiseptic is then applied with a gauze swab, such as a little iodoform powder, and successive layers are so treated with antiseptic after suturing.

4. **Closure of the Wound.**—This should be done by four or five through-and-through sutures, including skin, aponeurosis, and musculature. They may be of stoutest silkworm gut or of silver wire, and merely bring about apposition without undue tension. I have used silver wire in most cases: it is stronger, causes less irritation, is less likely to cut through, and it is not very painful to remove after the cut ends have been straightened out. At each end of the wound a small drainage tube must be inserted down to the sub-peritoneal tissue (*Fig.* 200). Although not always necessary, the musculature and the aponeurosis may have a few interrupted sutures of strong catgut nserted, but excessive suturing is to be avoided here. No sutures are put in the skin other than the deep through-and-through sutures. An antiseptic dressing is then applied.

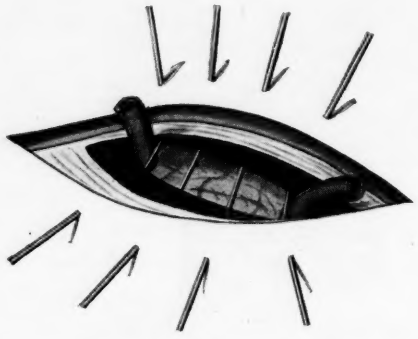


FIG. 200.—Method of closure and drainage of the wound.

SUBSEQUENT COURSE.

Natural defæcation is the rule, and frequently the wound heals quickly and securely. At the end of five days or a week there may be a slight fæcal discharge, which ceases during the next fortnight. Occasionally the wound breaks down completely, but after waiting two or three months the operation may be repeated with perfect success. In one of my cases a minute permanent fistula remained which discharged at times. The risk of temporary failure appears to be greater in the left inguinal region than elsewhere. It is here that the spur is thicker, being composed of the compressed portion of mesosigmoid; and in one case at this site end-to-end union had to be performed after an unsuccessful attempt at extraperitoneal closure.

The Mortality.—The death-rate is very low: in the absence of serious complications it should be well under 5 per cent. Of 38 cases recorded in the appended table there were 3 deaths, each with complications—previous gangrene of the bowel, cancer in the pelvis, and previous perforative peritonitis. Enterectomy by a two-stage method—Paul's method—is admittedly the *safest* for the large bowel, especially with intestinal obstruction, even with preliminary cæcostomy. If a comparatively safe and satisfactory method of extraperitoneal closure of the resulting artificial anus is added, the two combined have a lower mortality than other procedures, and are applicable to many conditions, as shown in the following table.

OPERATIONS FOR EXTRAPERITONEAL CLOSURE OF ARTIFICIAL ANUS.

PRIMARY CONDITIONS	AGES	NO.	REMARKS
	Years		
Succesful (34)			
Cancer of rectum	41	1	—
Cancer of colon	46–64	11	One double simultaneous closure
Cancer of sigmoid	35–74	5	Patient, age 74, was well three years after
Sigmoidectomy for prolapse, rectal and uterine	60	1	Permanent minute fistula
Irreducible intussusception ..	1 7/12–11	3	*See* text.
Sigmoiditis	60–77	3	One with large hernia, and bowels acted naturally after six years
Sigmoiditis (sigmoidectomy) ..	37	1	Second operation successful
Volvulus	40	1	—
Sloughing following appendicitis	21–30	2	Operation had to be repeated in one
Fæcal mass, peritonitis ..	50	1	—
Intestinal obstruction	47–56	2	In one, colostomy done at sea
Traumatic colostomy	—	3	War injuries
		34	
Unsuccessful (4)			
Cancer of rectum	46	1	Extraperitoneal a failure, successful end-to-end union
Gangrenous intestine	33	1	Death
Cancer of colon	70	1	Pelvic cancer present
Cancer of sigmoid with perforative peritonitis	65	1	Death two days after: bronchitis
		38	

Total Deaths, 3: one with gangrenous intestine, one with secondary cancer, and one with cancer and perforative peritonitis.

APPRECIATION.

The disadvantages are the loss of time and the two stages of operation, but these disadvantages are small compared with the gain in life. In rather more than half the cases the artificial anus had been required for *cancer* with obstruction; in one of these there had been perforation with peritonitis, and in another the cancerous mass burst at the time of the initial operation. The oldest patient was 74, with cancer of the sigmoid, and she is still well three years after the extraperitoneal closure. The youngest patient was 1 year and 7 months old. Three cases of *irreducible intussusception* in children have been treated by a two-stage enterectomy, with drainage of the upper bowel by tube or by lateral anastomosis, followed by an extraperitoneal closure of the artificial anus, all successfully; I submit, therefore, that the method is safer than primary excision or excision of the intussusceptum. There were 4 cases of *sigmoiditis*; in 2 of these colostomy had been done for supposed cancer with obstruction, the tumour disappearing subsequently. In one of these a very large hernia at the site of the colostomy was cured at the same time as the extraperitoneal closure was done, and it is of interest that the bowels act

naturally, although for six years all fæces had passed through the artificial anus. A third was a two-stage resection for diverticulitis which required a second, and successful, attempt at closure of the artificial anus. In one patient *two artificial ani* were successfully closed at one time, inguinal and left lumbar, the inguinal anus having been made for obstruction, but below the growth in the splenic flexure. The cure of artificial anus of the *small intestine* has proved to be as satisfactory as that of the large, after preliminary treatment of the excoriated skin, in which the 'button-valve' has played a valuable part.

Although the method was published over thirty years ago and I have employed it since 1898, there have been few references to it in literature. The closure of colostomy openings was discussed at a meeting of the Royal Society of Medicine in 1917. Several speakers mentioned the success of the extraperitoneal method, but the opener of the discussion said it left the gut outside the peritoneum and with a narrowed lumen, that the patients generally have chronic constipation and a weak place in the abdominal wall which requires the use of a support.[4] Properly performed, however, only the closed artificial anus is extraperitoneal and the gut is within the peritoneum. None of my patients has had a weak place in the abdominal wall, and none has worn a support, even when, as in one case, a large hernia the size of two fists was cured at the time of extraperitoneal closure.

Sinclair White advocated the method very strongly in the *Lancet*, and rightly remarks: "The one factor which makes the operation so much safer than resection of the bowel is that the mesenteric side of the colon with its vascular and nerve-supply is not interfered with."[5] Four years previously he wrote: "All the fatalities I have had in my private practice occurred in cases where the single-stage operation was done, and I am so firmly convinced of the superiority of the two-stage procedure that I have employed it exclusively for the last five years".[6] His experience was supported by Ernest Finch.[7] Robert C. Coffey[8] has reported a method of extraperitoneal closure which in all essential respects is similar to that described and practised by Greig Smith, whose memory we revere and whose method we would like to endorse and perpetuate.

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SARCOMATOUS PERMEATION OF THE INFERIOR VENA CAVA AND RIGHT SIDE OF THE HEART.

By H. J. B. FRY and C. E. SHATTOCK, London.

This case of intravascular sarcomatous growth is worthy of record not only on account of the length and continuity of the growth within the vascular system, but also for its unusual relation to the heart and vessels. As will be seen, the growth entered the inferior vena cava at its origin from the common iliac veins, traversed its whole length, forming a solid cast of the vessel, went through the right auricle and orifice of the tricuspid valve and the cavity of the right ventricle, and passed out through the pulmonary valve into the pulmonary artery as a solid cord. Portions here became detached and formed emboli in the lungs. The length of the growth within the vascular system as a solid mass was not less than 53 cm., with an average diameter of 5·5 cm.

This extraordinary growth formation must have been in existence for some months prior to death; yet it did not cause very severe vascular symptoms apart from œdema of the lower part of the body, in spite of the almost complete blocking of the inferior vena cava and right side of the heart, permitting the passage of only a thin peripheral layer of blood.

NOTES OF THE CASE.

M. C., age 19, primipara. Admitted Nov. 12, 1924, under Mr. Shattock.

History.—The patient was delivered of a full-term child four months previously without the aid of instruments. The child weighed 7 lb. 6 oz. During the last six months of pregnancy the patient was numb on the left side from just above the hip down to the knee, but no numbness was present elsewhere. On getting up ten days after labour, she complained of patches of numbness on sitting down, and there was numbness in the right leg from hip to knee, but none at all on the left side. The patient at this stage could not walk properly, being 'drawn over' on the right side and having difficulty in sitting down. Four weeks after her delivery she complained of severe pain in the lower part of the back, and her temperature was over 100° F. For the three months preceding admission to hospital she had complained of severe pain in the back, aching in character, relieved by standing up. The numbness in the right lower limb extended to the foot, but varied in degree. She complained of aching in the right leg, especially on walking, but no pain was felt in the left leg. There had been marked constipation for the past four months, but not previously. She had been getting thinner and had lately some difficulty in micturition.

On Admission.—When admitted to hospital on Nov. 12, 1924, the patient looked pale and thin. She walked with the right knee flexed and

the right heel raised off the ground, owing to pain. The movements of the right lower limb were somewhat weak. Objective sensation was normal in the right leg and thigh. The left lower limb showed no muscular weakness or sensory change. There was anæsthesia of the perineum both to pinprick and cotton wool, the area of anæsthesia extending down the upper part of the back of the right thigh. Knee-jerks present; plantar reflexes present and flexor in type.

Pelvic examination demonstrated a large hard mass projecting forwards from the sacrum, to which it was attached by a broad base. The mass extended more to the left of the middle line than to the right. Its upper border could not be reached. X-ray examination showed a large irregular shadow of the density of bone superimposed on the sacral promontory.

Laparotomy revealed a large hard tumour growing from the sacrum, extending widely upwards and laterally. A small piece of the growth was removed by a perineal incision for histological examination, and radium was inserted through this incision. The pathological report showed ossifying chondroma without malignant changes in the portion removed.

Subsequent Progress.—The patient developed urinary incontinence. She suffered with pain in the back and sharp pains in the right foot, especially on movement. Increasing weakness of the lower limbs and pain in both legs kept her bedridden, both legs being hyperæsthetic. An irregular pyrexia occurred at times.

Towards the end of her life enormous œdema of both lower limbs and vulva developed. The œdema later involved the abdominal wall. Multiple nodules of growth in the subcutaneous tissues of the buttocks and hips appeared, and a large growth was visible. The patient lived for several weeks in this condition of marked œdema, and died finally with uræmic symptoms twelve months after the birth of her child and eight months after first coming under observation.

Pathological Reports.—The Wassermann reaction upon the blood was negative. The piece of tissue removed at operation showed cartilaginous tissue with some areas of calcification and osteoid tissue, but there was no definite evidence of sarcomatous change, and the growth was diagnosed as an ossifying chondroma.

Post-mortem Examination.—This showed marked œdema of the lower extremities and the lower part of the abdomen, and there was some swelling of the face and neck. On incision, the subcutaneous tissues of the abdomen poured out œdematous fluid, but there was no ascites. In the region of the sacrum was a large mass lying behind the peritoneum, almost filling the cavity of the pelvis and extending up into the lumbar regions. The growth arising from the sacrum was smooth, firm, with a slightly nodular surface, and its limits could not be defined. It was covered by cartilage below in the pelvis, but above in the region of the psoas muscles and in the inguinal regions the growth was gristly, with calcareous areas.

The inferior vena cava was greatly distended and at first hardly recognizable, being a rounded semi-solid pillar extending from the junction of the common iliac veins to the lower surface of the diaphragm. It appeared to be completely thrombosed. The common iliac vessels on both sides were

involved and compressed by the growth. The renal veins were not invaded. The lumen of the inferior vena cava was occupied by a mass of firm, whitish, gristly consistency which extended through the right auricle into the right ventricle and passed out as a solid cord through the pulmonary valve into the pulmonary artery (*Fig.* 201). The growth had a vermiform appearance, with its surface broken up by small gyri and sulci, giving it a nodular glistening character. On longitudinal section the mass is seen to be composed of growth and organized thrombus compacted into a solid mass, with reddish clots in a few central areas. The superior vena cava was free from growth and contained post-mortem clot.

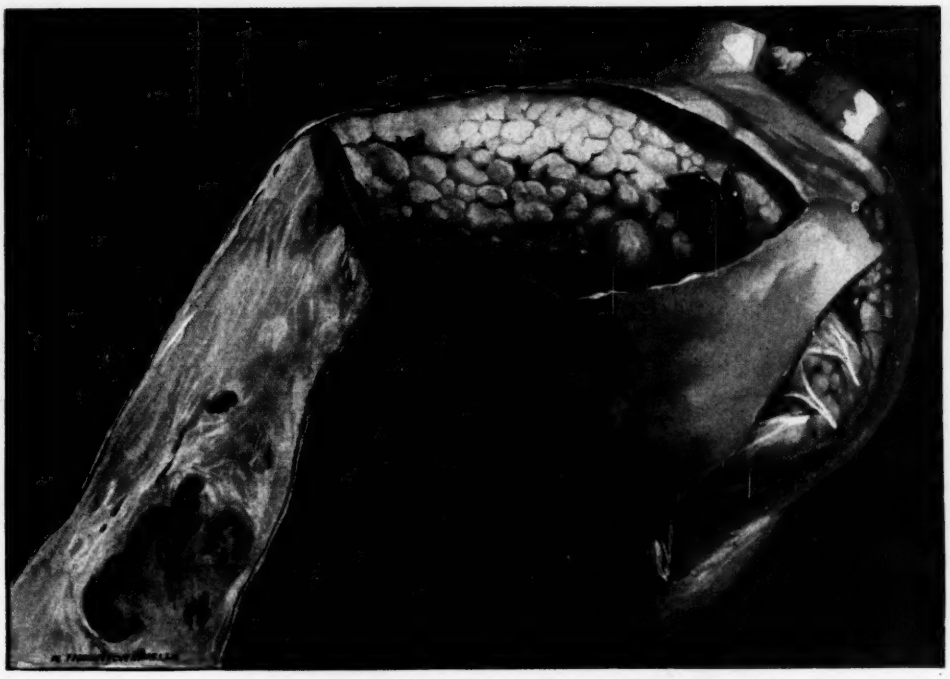

FIG. 201.—Vena cava and right heart filled with growth and thrombus.

The pleural cavities contained a considerable quantity of clear fluid. The lungs were partially collapsed, and the lower lobes on both sides contained a few small rounded whitish nodules of growth in their substance.

The thoracic aorta was rather small in calibre, and the abdominal part was compressed. The lumbar veins were distended. The liver showed changes due to marked passive venous congestion, with fatty degeneration. The spleen was small and tough. The mucous membrane of the stomach showed œdema and superficial areas of necrosis. The ureters were compressed by growth on both sides as they crossed the brim of the pelvis. They were dilated above this point, and the kidneys showed the changes of early hydronephrosis. The adrenals were healthy. The uterus and appendages were normal.

Death was due apparently more to uræmia than to the growth or its immediate effects.

MICROSCOPICAL EXAMINATION.—

1. *Sacral Growth.*—The tissue shows a fairly dense fibrous stroma interpenetrated with more or less dense aggregations of spindle or polymorphic cells resembling young fibroblasts, forming a meshwork. Here and there throughout the tissue are cartilaginous areas undergoing varying degrees of calcification. The cells described above are of fairly regular size. The nuclei are not abnormal in appearance and show few mitoses. This tissue varies from that taken at operation, in the smaller amount of cartilage present and the increase in the fibrocellular tissue and in the areas of calcification. These differences may obviously be due only to the nature of the samples taken in each instance.

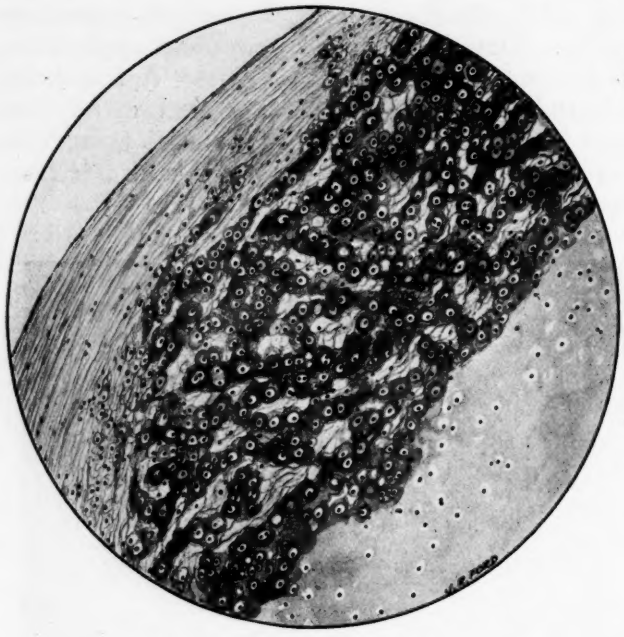

FIG. 202.—Section from growth in vena cava.

2. *Growth in the Inferior Vena Cava.*—In this tissue, immediately beneath the wall of the vessel is a layer of well-formed cartilage cells, separated from the wall here and there by a thin layer of fibrin and red cells. The cartilage cells are more regular than in the sacral growth, and show minute traces of calcareous deposit only at some points. The fibrocellular tissue is much less evident, and is seen only in relation to the cartilage or in small isolated nests of sarcomatous cells. Internal to this layer

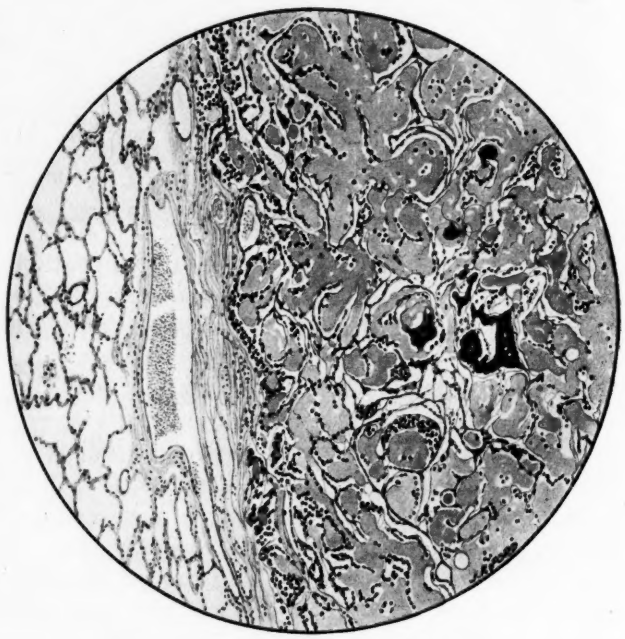

FIG. 203.—Section from nodule in lung.

is an almost structureless thrombus, partially organized, in which can be recognized with difficulty a fibrin network and red cells. This is partly penetrated by the sarcomatous cells (*Fig.* 202).

3. *Lung Nodule.*—This contains no true cartilage, but is composed chiefly of condensed masses of white fibrous tissue embedded in the alveoli of the lung, with rather numerous foci of calcification towards the centre of the nodule, where the fibrous tissue is less compact, forming wide meshes. At the periphery of the nodule are grouped the sarcomatous cells. Here they are more definitely spindle-shaped or form small syncytial masses, and are relatively numerous. There is a capsule of condensed lung tissue (*Fig.* 203).

REPORTS OF PREVIOUS CASES.

An intravenous and intracardiac growth of such size and continuity seems rarely to have been reported. The following are the only cases of similar character which we have been able to discover. Sir James Paget[1] in 1855 described a malignant enchondroma of the testis, which permeated the lymphatics of the spermatic cord. A secondary mass in front of the inferior vena cava pierced the latter and formed nodules on its inner surface and in many of the larger branches of the pulmonary arteries, producing secondary deposits in the lungs. This tumour was re-examined forty years later by Kanthack and Pigg[2] and considered by them to be a columnar-celled carcinoma, but was again examined by Nicholson[4] and regarded by him as an embryoma, derivatives of all three embryonic layers being present (*Case* 60).

Kanthack and Pigg[3] gave a report of another case of columnar-celled carcinoma of the testis in which the lumbar glands were invaded. From this a mass protruded into the inferior vena cava, extending upwards within its lumen, sending a lateral offshoot into the right renal vein. On the tricuspid valve was a dendriform mass extending through the right auricle into the inferior vena cava and into the right ventricle. The lateral branches contained small cysts filled with clear fluid. The authors consider that a fragment of the intravenous growth had become separated and grafted upon the tricuspid valve.

Parkes Weber[5] described a case of bilateral malignant adrenal carcinoma, in which there was thrombosis extending into both iliac veins below, and upwards to the right side of the heart, where it terminated in a rounded mass which projected into and partially filled the right auricle. This specimen is in the Museum of the Royal College of Surgeons, London, under catalogue number 2033.1. Examination of the clot at this point showed that it was infiltrated by the same form of malignant growth. There were secondary nodules in the lungs and liver. The patient died of uræmia.

French[6] reported a case of spheroidal-celled carcinoma of the left kidney which spread into the left renal vein and thence into the inferior vena cava, causing progressive thrombosis with malignant infiltration of the clot. This process continued up to the formation of a large polypoid mass in the right auricle, causing a tricuspid stenosis of the ball-and-socket type.

DISCUSSION.

The unusual length and continuity of the growth in the heart and in a vessel of such wide calibre are the chief features of pathological interest. Sampson Handley showed that the permeation of the lymphatics by carcinoma occurs chiefly in vessels of 40 to 50μ in diameter, avoiding the larger trunks where the force of the stream may be strong enough to sweep the cells away as emboli. The hard and elastic nature of the tumour in this instance probably aided its extension along the vena cava and prevented its being broken up by the blood-stream on its entry into the lumen of the vessel, which the thinness of the wall made easy. The comparatively slow character of the growth and its relatively low cellular content not only assisted the great extension of the growth, but also permitted the gradual re-adjustment of the vascular mechanism. The enlargement of the veins of the anterior abdominal wall, to which Weber has drawn attention in thrombosis of the inferior vena cava, was not a feature in this instance. It is remarkable that in this case, and in those quoted above, the patient lived so long and died finally of uræmia.

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THE SURGERY OF THE JEJUNUM.

(Being a Hunterian Lecture delivered at the Royal College of Surgeons of England on February 3, 1926.)

By JOSEPH E. ADAMS, London.

To the anatomist the jejunum is the upper two-fifths of the small intestine; to the surgeon it is that portion which begins at the duodenojejunal flexure and does not descend into the pelvis. It might appear that its surgery need hardly be considered apart from that of the rest of the small intestine; but I think a separate consideration of it may be worth while, especially in view of the fact that of recent years the operation of gastrojejunostomy has brought in its train troubles which affect the jejunum alone.

TRAUMATIC LESIONS.

Although the jejunum is nowhere protected by any covering but muscle, the incidence of small-intestine rupture is about equal in both jejunum and ileum. It may be that the relative emptiness of the jejunum affords some immunity. If it is accepted that the three possible mechanisms, a crush, a tear, or a burst, are the factors responsible for intestinal rupture, I think we may take it that the last very rarely affects the jejunum. Sir James Berry[1] found that 90 per cent of cases of intestinal rupture occurred in males, mostly between the ages of twenty and fifty. Undoubtedly fixed portions of the intestine are most susceptible to tearing. The commonest cause of intestinal rupture is a direct blow on the abdominal wall by some object which involves only a small area but will push its way on so that anterior and posterior walls temporarily come into contact. What happens to the bowel between them depends on its fixity and its state of distention. If the rupture occurs at, or close to, the beginning of the jejunum, I think it is right to infer that the lesion is a tear. Massie,[2] in reviewing 34 cases at Guy's Hospital involving all parts of the bowel from stomach to rectum, found nearly 50 per cent in the jejunum, and half of these again were within eighteen inches of the duodenojejunal flexure.

The clinical aspect of intestinal rupture has been very closely studied, and no description of the physical signs is needed here; but an inspection of hospital statistics does reveal the fact that many cases are watched for hours after admission before operation is undertaken. The most important point to be considered is the nature of the accident. If the abdomen has been struck by the hoof of a horse, the pole of a cart, the dumb-iron of a motor car, this in itself may often be sufficient justification for abdominal exploration. The prognosis can only be hopeful where operation is for rupture of the intestine and not for perforative peritonitis. Marked rise in the pulse-rate, recurrent vomiting, diminution of liver dullness, and abdominal rigidity are all

late signs. Local hyperæsthesia should be a sign of value, but, unfortunately, it is often unreliable on account of bruising of the abdominal wall. If no improvement takes place within two hours of the injured patient's being put in a well-warmed bed, it is highly probable that internal visceral damage is present, and the abdomen should be explored.

The prognosis of rupture of the jejunum is governed chiefly by the state of emptiness or otherwise of the bowel, and by the time which elapses between operation and the receipt of the injury. The time of the last meal should therefore be ascertained.

It is remarkable that in not a few cases a very slight fall on the flat abdomen has been responsible for rupture of the intestine. In most of these the accident has occurred shortly after a full meal. These are probably examples of bursting of the gut owing to its sudden compression at two or more points (Hain).[3]

The method of suture should be such as will avoid serious narrowing of the bowel lumen, and in cases of multiple or extensive ruptures resection may be necessary.

RESECTION OF SMALL INTESTINE.

This brings one to a consideration of the amount of small intestine which can be removed with safety. In this connection Pirrie Watson[4] quotes a case, recorded by Denk in 1910, where Bremer removed 17 ft. 9 in. for strangulated femoral hernia. The patient, who was a woman of 61, made a good recovery, and she lived for two and a half years, ultimately dying of marasmus. The only obvious defect in her metabolism during this period was a slight failure of fat absorption. It is clear, therefore, that the jejunum is a portion of the bowel which can be dispensed with, and, experimentally, Flint[5] found that 50 per cent of the small intestine could be safely removed from dogs. They lived on indefinitely in good condition, and the resultant early diarrhœa soon ceased. Compensatory hypertrophy of the remaining mucous membrane takes place, but there may be evidence that fats are not absorbed, and dietetic error may quite easily lead to enteritis and marasmus. Fractional resection is more readily tolerated, even up to 75 per cent of the small intestine.

Removal in the human being of small bowel up to two-fifths of its length makes little difference to metabolism, but I have been unable to find any case on record where the portion removed consisted of the whole jejunum. Most of these resections involve ileum rather than jejunum, since the type of lesion calling for such drastic surgery is likely to be a gangrenous strangulated hernia. The absorptive surface of the jejunum is greater, because the valvulæ conniventes are more pronounced in the upper intestine, and for this reason perhaps jejunectomy is more likely to upset metabolism than ileectomy.

DIVERTICULA OF THE JEJUNUM.

I do not propose to discuss the etiology of these protrusions or pockets in the wall of the jejunum, and I believe the distinction between 'false' and 'true' is largely an artificial one. Obviously, as any such protrusion increases in size it will tend to stretch the muscular coat, and this may lead to atrophy;

thus we get the change from a true to a false diverticulum, from so-called diverticulitis to sacculation. As one would naturally expect, this condition is rare in the jejunum, and the probable explanation is the relative absence of a high or persistent intra-intestinal pressure. Writing in 1921, Hunt and Cook,[6] quoting Case,[7] stated that from 1854 to 1920 only 17 certain cases had been recorded of diverticula in the small intestine; but Helvestine,[8] two years later, refers to 27 cases of false diverticula in the jejunum alone. Probably, therefore, the increasing frequency of opaque-meal examinations is bringing more cases to light. How far they are likely to be responsible for symptoms is problematical. Epigastric pain with tenderness and nausea are alleged to be attributable in some cases to their presence. My own clinical acquaintance with diverticula is limited to the duodenum and sigmoid colon.

The most striking example of jejunal diverticula was recorded by Braithwaite[9] in 1923. This patient was a man of 54, giving a history of dyspepsia for over thirty years. He had pain and nausea about one hour after food, with noisy flatulence. The symptoms were relieved about two hours later by the evacuation of wind, either by the mouth or anus. Vomiting took place three or four times a week, and on one occasion he had a profuse hæmatemesis. His symptoms before operation were attributed to tuberculous peritonitis. The jejunum showed no fewer than fifty diverticula along its mesenteric border, and presumably all the coats were involved. The highest was situated at the duodenojejunal flexure, and their size varied from that of a tangerine orange to a pea. Four feet of the bowel were successfully excised, and no other diverticula were discovered in the abdomen. The specimen is most remarkable, and affords abundant scope for theorizing as to the causation of such diverticula.

THE JEJUNUM AS THE SEAT OF INTESTINAL OBSTRUCTION.

The higher the obstruction, the more urgent the symptoms of pain and vomiting. Jejunal obstruction, if unrelieved, rapidly terminates fatally. This portion of the intestine may be implicated in any of the ordinary forms of obstruction, whether it be internal hernia, adhesions, intussusception, volvulus, or due to new growth. Naturally its relationship to the duodenal fossæ makes it almost certain that an internal hernia in this region will involve the jejunum. Of the other causes of obstruction probably adhesions are the commonest. Intussusception is rarely met with in this part of the small intestine. That volvulus may occur apart from the presence of initial adhesions or abnormal mesenteric arrangements appears certain. Irregular peristalsis must in such cases be responsible, and this may be caused by injudicious or excessive feeding, or the misuse of aperients. Inherent irregularities in the muscles of the bowel wall may be a necessary factor, and Carey, quoted by Tees,[10] has shown that in some animals the musculature of the intestine does not consist simply of an inner circular coat and an outer longitudinal one. Instead of this the inner coat may form a closely-wound spiral and the outer a more elongated spiral. I am not aware that this state of affairs has ever been found in a human subject, but irregular peristalsis of such muscular coats might easily be responsible for volvulus and consequent obstruction.

I operated upon a case involving some eight inches of the jejunum a few years ago, but unfortunately I have been unable to find the specimen. The gut was gangrenous and the affected bowel was resected, but three or four days later the patient died for no very obvious reason, since the anastomosis was perfect and there was no peritonitis. Probably a temporary jejunostomy should have been added to the resection to save the patient from toxæmia, and the argument in favour of this proceeding may now be considered.

The question of first importance with regard to obstruction of the small intestine is the precise explanation of its high and rapid mortality. Many investigations have been made to elucidate this problem, and the researches of Stone, Whipple and Bernheim[11] are no doubt familiar. It is generally admitted that death resulting from acute obstruction is due to the absorption of toxic material developed within the obstructed bowel. From artificially obstructed loops in dogs the poisonous fluid may be abstracted. When it is injected intravenously into healthy animals death ensues with symptoms precisely similar to those presented by the dogs with obstructed loops. Such animals exhibit, according to Stone,[12] "a marked and rapid fall of blood-pressure, temperature disturbances, excretion of large amounts of fluid into the intestinal canal, with vomiting, diarrhœa—sometimes bloody—and fatal shock."

A marked engorgement due to vascular dilatation of the mucosa of the digestive tract, especially the duodenum and jejunum, was a feature of the autopsy findings.

The source of these toxins and their chemical nature is still the subject of controversy. Whipple[13] himself has maintained that the poison is a product of the mucosal wall, especially of the duodenum, but many workers, notably the Dagstedts,[14] have disputed this conclusion, and have found that bacteria and the results of bacterial activity are necessary to the fatal issue. Closed loops of bowel freed from bacteria are quite compatible with health. Protein substances are acted upon by bacteria in closed loops not rendered free from organisms, and of all the products proteoses appear to be the most toxic. Such toxins must be absorbed and reach the general circulation in order that lethal results may follow, and a moot point is whether the mucous membrane of an obstructed loop can or cannot absorb these substances. Whipple maintains that it cannot, but others hold that the circulatory changes in the mucosa, with tissue necrosis due to bacterial action, do cause absorption to occur. Stone[15] suggests that the toxin, or toxins, present in the obstructed gut, bearing a resemblance to histamine, themselves cause increased tone and peristalsis in the bowel. Vomiting does not remove the toxin, and as more is produced its absorption is increased. If the bowel were unobstructed it would be hurried along and the lethal event avoided.

Of the practical value of jejunostomy in obstruction I shall speak later, but the experiments of Wilkie[16] deserve some consideration. He very reasonably criticizes the foregoing experiments because the toxins recovered from the obstructed bowel were injected intravenously. The crucial question is the degree of absorption which takes place in the bowel above the obstruction. In eight animals Wilkie injected into unobstructed bowel the fluid contents from the small intestine of animals dying from obstruction. In

none were any toxæmic symptoms seen within twenty-four hours. Two of the animals, however, developed an acute enteritis. He concludes that damaged or devitalized mucosa is essential for toxic absorption. Stone thus explains the lack of lethal effects in Wilkie's experiments. But Wilkie also failed to produce the lethal effects of pure obstruction even when he introduced the toxic material into bowel above an obstructed loop so that peristalsis could not pass the toxic material along and thus prevent absorption and toxæmia. Wilkie concludes, and I think with reason, that paresis of the jejunum is a most important contributory factor in toxic absorption, and this may exist and persist even when obstruction is successfully relieved by operation. Evacuation of the bowel contents is one important factor in recovery, but restoration of peristalsis is also essential. The jejunum is constructed for absorption, and it is probably right to speak of its area as the " poisonous proteose level " in obstruction.

On the clinical side the obstruction due to an impacted gall-stone is a variety particularly liable to lead to this latent toxæmia, and not infrequently the obstruction is very easily dealt with and yet the patient dies. Presumably fatal absorption takes place because of failure of peristalsis, and the disease would possess an even higher mortality than it has if the impaction occurred in the jejunum rather than the ileum, where such a stone is more commonly arrested.

NEOPLASMS AND CYSTS.

Neoplasms.—A careful perusal of the literature confirms one's personal experience that new growths rarely affect this portion of the intestine. It is certainly not much subjected to the irritation of fæcal matter, and since this is commonly regarded as one of the factors in the production of epithelial malignant disease, it is not strange that primary carcinoma of the jejunum should be almost a pathological curiosity. The same is not true of sarcoma, and probably half the intestinal sarcomata arise in the small intestine, affecting jejunum and ileum about equally. Such a tumour has several times been observed as the starting-point of an intussusception of the jejunum. According to Goldstein[17] these growths are usually lymphosarcomata, but spindle- and round-celled tumours are also found, and they may contain melanin. They may form polypoid tumours or be infiltrating in type.

All benign tumours of the small bowel are rare. A pure fibroma of the jejunum, the size of a fist, was removed by Miller in 1915, and this is figured in an article on benign intestinal tumours by King,[18] who reviewed the literature and found that, of the simple tumours of the intestinal tract, myomata were the most common; then came lipomata, adenomata, and fibromata. Out of 119 cases, only 8 concerned the jejunum without question. Tumours of the ileum were at least three times as common.

Cysts.—Apart from the cystic degeneration of tumours, there are two types of cyst which call for our attention in connection with the surgery of the jejunum—cysts of the mesentery, and cysts of the intestine itself.

Mesenteric cysts have been known ever since the sixteenth century, but the number recorded is said not to exceed 300 even up to the present time (Higgins[19]). Their etiology is still uncertain, and several classifications have

been attempted. We may at once dismiss those which are parasitic, and the dermoid cysts, leaving us with the so-called 'true' mesenteric cysts, most likely of developmental origin. It was assumed at one time that dilatation of lymphatic spaces was responsible for these cysts; that they were comparable to the single or multiple cysts seen in the necks of young children, to which the name cystic hygroma is applied. I see no reason to abandon this as one of the possible causes, but it is also probable that they may arise from Wolffian remains, or as diverticula from the bowel. A persistent portion of the vitelline duct may at times be responsible. Some of these cysts may be traumatic in origin. As regards the mesentery of the jejunum, it is a rare site for such cysts, and they are undoubtedly most common near the termination of the ileum. A case of exceptional interest was recorded by Makins[20] in 1911 of a multilocular cyst in a child of 6, which caused acute obstruction by pressure on a portion of the jejunum. From infancy the child had been subject to attacks of abdominal pain with occasional vomiting. The operation was performed for urgent symptoms of six weeks' duration, and three inches of jejunum were flattened out like a band on the surface of the cyst. The contents consisted of chocolate-coloured fluid in one sac and of colourless fluid in the others. Before operation a large tumour could be felt in the umbilical region. Excision of the cyst with the implicated bowel, and axial anastomosis, were successfully performed. Makins assumed that these cysts were examples of lymphangiectasis, but the wall of his cyst had no endothelial lining, and consisted of fibrous tissue with some non-striped muscle fibres. The presence of these muscle fibres goes some way to suggest an enterogenous origin.

A type of cyst of very great interest and considerable rarity is also met with about the jejunum, but on its antimesenteric aspect rather than along its concave border. I refer to the disease called cystic pneumatosis. An admirable description of this was given by Nitch[21] in his presidential lecture before the Surgical Section of the Royal Society of Medicine in 1923, and he is responsible for the statement that " the jejunum is frequently involved as well as the ileum, but it is rare to find cysts on this portion of the bowel alone ". Only about 90 cases in all have been described, and the etiology of these gaseous cysts is entirely speculative.

INFARCTION OF THE JEJUNUM.

In my researches into the literature I have so far failed to find a case of infarction limited to the jejunum. A consideration of the blood-supply of the bowel would naturally lead one not to expect such a lesion.

CONGENITAL OCCLUSION OF THE JEJUNUM.

In a review of 392 cases of congenital atresia of the intestine, from pylorus to anus, Davis and Poynter[22] found that its incidence in the jejunum and ileum was about equal, so that the condition is not specially attributable to errors in development about the vitelline duct or the ileocæcal valve. One-third of the cases affected the duodenum. They describe a case of multiple strictures limited to the jejunum in a child forty-eight hours old, which could

be treated only by enterostomy. Death ensued six hours later. The post-mortem revealed no atresia elsewhere. The mesenteric arteries were normal in distribution, but opposite all the atresic portions of bowel the walls of the vessels were sclerosed, and the terminal branches of the arteries were occluded by an overgrowth of cells apparently derived from the endothelium.

ULCER OF THE JEJUNUM.

Any and every variety of ulcer has been found in the jejunum, but that which we are most accustomed to meet with is ulceration following gastro-jejunostomy. This is an example of man-made disease. The question of greatest importance is whether peptic ulcer can occur in the jejunum apart from this short-circuiting operation, which allows gastric juice to come in contact with the jejunum before neutralization has occurred.

This question has been admirably discussed by Richardson[23] in 1922, and Patterson Brown[24] in 1924. The former deals only with ulcers of the jejunum; the latter with those which are 'simple' and affect jejunum and ileum. Van Roojen[25] in 1909 described 3 cases of what he regarded as peptic ulcer of the jejunum without gastro-enterostomy, and the importance of his observation centres round the word 'peptic'. Richardson recorded two cases of his own. In one the ulcer was only twelve inches below the end of the duodenum; in the other it was located about the middle of the jejunum. He analysed 10 other cases from the diagnostic point of view, and in these 12 examples of ulcer the adjective 'simple' was fully justified: though syphilis could not be absolutely ruled out, for the results of a Wassermann reaction were not recorded, and only one was examined microscopically for the spirochæte of syphilis. Perforation occurred in 10 of these 12 cases. Several of them bore a distinct resemblance to the ulcer commonly met with in the stomach, and in 7 the ulceration was obviously chronic. One was annular, but dysentery, malignant disease, and tuberculosis appear to have played no part in the causation of any of them. Typhoid, uræmia, and simple distention were also excluded. Richardson, however, objects to the term 'peptic' on the ground that we should have to accept the existence of a digestive ulcer in an alkaline medium, and their very rareness makes this theory improbable. In only two of the cases was there coexistent ulceration of the stomach or duodenum. There are again other unclassified ulcers in the rest of the small intestine, so it would appear impossible to be more definite at the present time than to speak of these ulcers as simple, and for the most part chronic. Even in 1924 Patterson Brown was able to collect only 35 cases for both jejunum and ileum, so that we may assume that primary jejunal ulcer remains a very rare disease. It is quite otherwise with jejunal ulcer following gastro-enterostomy, and to this subject some attention must be paid.

Ulceration following Gastrojejunostomy.—This important sequel to an operation, which, to quote Mr. Sherren's[26] recent Bradshaw lecture, "has done as much, if not more, for the good of the human race than any surgical procedure", I have already referred to as a man-made disease. It is a most unfortunate blemish on the surgery of the stomach, and I do not think any surgeon of experience can claim that his patients are immune from it. That

it is an artefact of gastric surgery is supported by my argument with regard to simple jejunal ulcer. That special precautions taken whilst doing the anastomosis have reduced its incidence goes further to prove that the surgeon is often responsible. The use of absorbable suture material appears to be the most essential of these precautions, and Mr. Sherren has advised against the use of a clamp on the jejunum, which is a luxury rather than a necessity for the surgeon. It may perhaps be responsible for damage to the wall of the jejunum and thus possess a hidden danger for the patient.

It is usual to speak of gastrojejunal or anastomosis ulcer, and jejunal ulcer proper, but there is no doubt that the anastomosis ulcer is the commoner, and Walton[27] would have us believe that even when the ulcer is found in the jejunum it has always spread downwards from the anastomotic line. Sherren does not agree with this; neither do I, for I have operated on a perforating ulcer of the jejunum several inches from the suture line and not connected with it by fibrous tissue. After all, any one surgeon's experience of this lesion is not great, and it is perhaps true that the more gastro-enterostomies he does the fewer post-operative ulcers does he see—or, shall I say, cause.

Nine cases of this lesion have come under my immediate care, and, of these, 6 should be classed as gastrojejunal, 2 as jejunal, and 1 presented a gastro-jejuno-colic fistula. In 5 of them the gastro-enterostomy was performed by myself, and this represents roughly an incidence of 2 per cent. I am in the habit of using clamps on both jejunum and stomach. Up to 1917 I used silk suture-material, and also removed some mucous membrane from the jejunum. This I believe now to be undesirable, and there is something to be said for a separate stitch for the mucosa. Microscopical examination of anastomoses at various periods after the operation has convinced me that the average suture line—and I have sectioned those of several surgeons—is very irregular, and the time of healing must be very variable.

A matter which appears to me of the greatest importance in technique is the treatment of the mucosa. Moynihan[28] in 1914 advocated removal of an ellipse of mucous membrane from both stomach and jejunum, and other writers have followed the same lines. The more recent text-books on operative surgery urge economy in the matter of the mucous membrane. The object is to secure an immediate and relatively large opening and avoid a mere slit, but I do not believe this can always be done without running the risk of delayed healing. Better some swelling of the margins of the wound, and temporary blockage of the anastomosis, than failure of mucosal healing and exposure to erosion by acid secretion. How many surgeons realize the importance of the conservation of every bit of mucous membrane it is difficult to know, but Sherren[29] is quite clear on the point, and he does not remove any redundant mucous membrane. That of the jejunum always appears to be excessive, but nevertheless I believe it should never be excised. The microscopical section shown in *Fig.* 204 came from a case where the gastro-enterostomy was perfectly satisfactory from a clinical point of view, and the patient's death was unconnected with the peritoneal cavity. The suture material used was 00 iodized chromicized catgut, and the specimen was obtained from a post-mortem examination twenty-four hours after death. The anastomosis was performed sixteen days before the specimen was recovered

from the post-mortem room. To the naked eye there appeared to be perfect continuity of surface between stomach and intestine. The microscope, however, shows that there is in reality a mucosal ulcer, and the gap is filled up with fibrous tissue, in the deeper layers of which remains of mucosal cells can be identified. No mucous membrane was excised in this case either from the stomach or the jejunum, but it is evident that necrosis has occurred from the embrace of the suture, and this appears to be inevitable. The only way to diminish this loss of tissue would be to use interrupted stitches; but most surgeons will cling rather tenaciously to the hæmostatic suture; if it is interrupted its hæmostatic value is lost. Exactly how long the process of

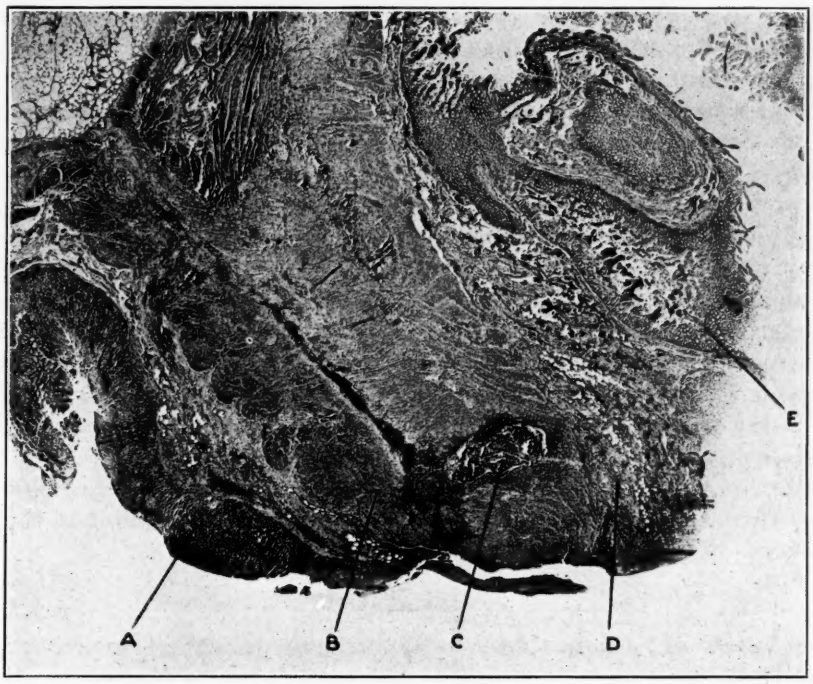

FIG. 204.—Shows pronounced gap in mucosa revealed only by microscopy. To the naked eye the mucosa of stomach and intestine appeared to be continuous. A, Mucosa of jejunum; B, Muscularis of jejunum; C, Unabsorbed catgut; D, Fibrous union of stomach and intestine; E, Mucosa of stomach.

final healing takes is, I think, an unknown quantity, but I should put it down at quite one month, and possibly two. For this reason a careful dietetic régime is rightly insisted upon for at least three months from the date of operation. Fibrous tissue is not proof against the attack of hydrochloric acid.

The two cases which I should classify as true jejunal ulcer in my practice have occurred in patients who started their unfortunate gastric career with a perforation of the jejunum. In both of them an anterior gastro-enterostomy was performed later. In one case the perforation was in the afferent loop, and the anastomosis was drawn out into a tubular connection between the stomach and the jejunum. The other jejunal perforation was a recurrent one, and I cannot say positively that there was no inflammatory thickening

along the line of the anastomosis. This perforation was in the distal limb of the jejunum an inch and a half below the junction with the stomach.

The clinical aspect of gastrojejunal ulcer is a rather difficult one. Not all patients upon whom gastro-enterostomy is performed for a positive lesion are at once converted to the eupeptic state. Some are, and their enthusiasm is very gratifying. Some convalesce slowly, but, nevertheless, they give a perfect end-result. Some resemble " seed sown on stony ground " and they relapse. Sherren, I think, has been a little too sweeping in asserting that a gastro-enterostomy patient who is perfectly happy for two years after the operation never gets trouble again, and does not run the risk of a gastrojejunal ulcer; but the majority of such patients will admit to having experienced periods of discomfort shortly after the operation. Gradually their symptoms acquire the characteristics commonly associated with duodenal ulcer. Hunger-pain is often a prominent feature. The only physical sign in peptic ulcer on purely clinical examination is tenderness on pressure. This, in the case of gastro-jejunal ulcer, is placed to the left of the umbilicus and a short distance above it.

Carman[30] gives the following points in connection with the diagnosis of gastrojejunal ulcer by means of X rays: (1) Retention from the six-hour meal; (2) Pain on pressure in region of first loop of jejunum behind the anastomosis; (3) Impaired peristalsis; (4) Ampulla-like distention in the region of the anastomosis; (5) Distortion of the stoma and pain on pressure; (6) Narrowing of the efferent limb of the jejunum near the stoma; (7) Dilatation of duodenum or afferent limb of jejunum; (8) A definite pocket retaining the barium is rarely seen. Since the last-mentioned sign is *the* crucial one in the X-ray diagnosis of any ulcer, it will be readily seen that a positive diagnosis by radiography is by no means easy to obtain. Certainly not all deformities of the jejunum after anastomosis point to an ulcer; but if the clinical signs suggest it, there is usually support for the diagnosis in the X-ray appearances.

JEJUNOSTOMY.

The Value of Jejunostomy.—Jejunostomy is an operation which is comparatively rarely done, but I shall endeavour to show that there are many conditions for which it may be useful, and there are several, I believe, where it is positively indicated. As a place for the reception of food the jejunum is quite satisfactory. Milk, eggs, and sugar poured in by means of a tube and funnel at four-hourly intervals will easily maintain the nourishment of the patient. At first the quantity administered should not exceed 6 oz., but in quite a short time the jejunum becomes accustomed to further distention, and 10-oz. feeds can be given. It appears to make little difference whether the milk is pancreatized or not. Such jejunal feeding is quite well borne after the first few days, and the majority of patients will cheerfully submit to a régime which permits neither fluid nor solid by the mouth provided they are thus rid of their pain. But the use of a jejunal fistula is not limited to the ingestion of food. Jejunal drainage is the key-note to the successful relief of intestinal toxæmia which we have already discussed in dealing with high intestinal obstruction. I shall therefore discuss the value of this operation from the aspects of both ingress and egress to and from the intestinal canal.

Technique of Jejunostomy.—It is clear that a modification of the Witzel gastrostomy, originally suggested by Moynihan, has much to recommend it (*Figs.* 205, 206). It is rapid, it can be done without risk of soiling the peritoneum, and the loop of jejunum selected requires very little anchoring to the parietal peritoneum. If a couple of stitches be used for this purpose at the top of the wound, it is usually quite enough. Perfect security of the tube itself is most important, and a self-retaining winged catheter is best, although it is a little awkward to introduce. If an ordinary catheter is used, it is well to cut off its tip so as to make the opening terminal and not lateral. It is a good plan to encircle the tube with a stitch as it is brought out through the rectus sheath, and also as it lies upon the surface of the skin, for if the tube is

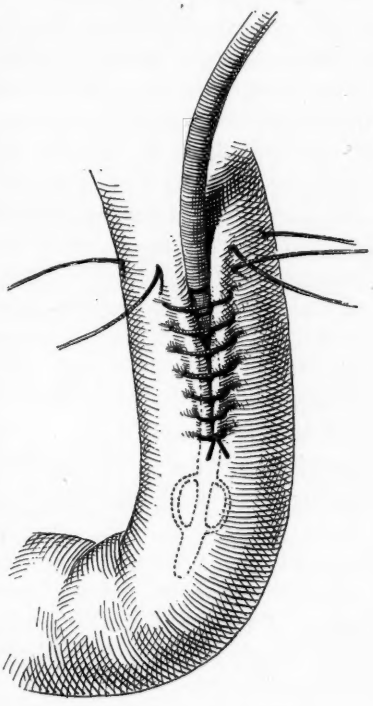

FIG. 205.—Technique of jejunostomy. Catheter within the lumen of the bowel. Note the anchoring peritoneal sutures.

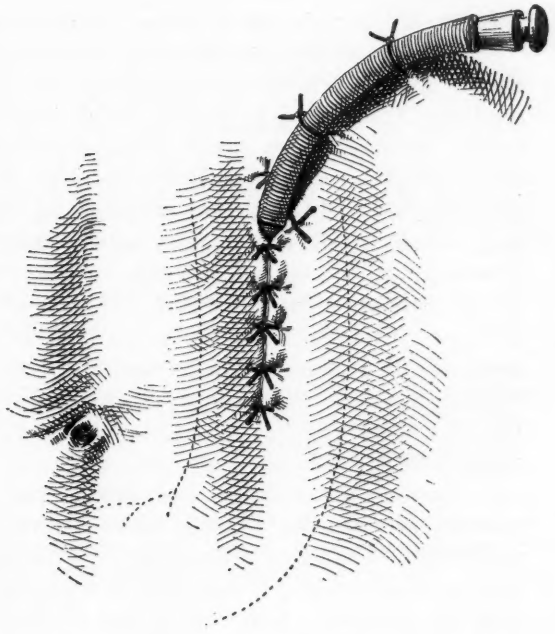

FIG. 206.—Technique of jejunostomy. Skin incision closed and tube anchored to the surface of the skin above.

dislodged during the first few days it will be well-nigh impossible to replace it. This operation can be carried out quite easily under local anæsthesia through the upper fibres of the left rectus, provided each layer is thoroughly infiltrated before division. If speed is essential owing to the poor condition of the patient, jejunostomy may be done through the mid-line, and even simple anchorage of a loop of bowel to the anterior abdominal wall with a catheter tied in will suffice, but there is a danger of leakage when the tube is removed. This hardly ever occurs if the Witzel technique is followed.

Jejunostomy as a Substitute for Gastrostomy.—This is the most obvious indication for jejunostomy. In my experience it is just as efficient as

gastrostomy, and easier to perform without risk of leakage. It is therefore appropriate as a means of feeding in cases of *carcinoma of the œsophagus* where the stomach is small and immobile, as well as in cases of *malignant disease of the stomach* itself that are unsuitable for excision or gastro-enterostomy.

In certain cases of *chronic gastric ulcer* there is a distinct field of usefulness for jejunostomy. It is the only method of continuous feeding whereby the stomach can be given absolute rest and the nutrition of the patient maintained even whilst at work. For this reason it has been recommended for bleeding ulcers of the stomach and duodenum. It is in connection with inoperable ulcers that I have found it most useful. Moynihan[31] has made use of this method of feeding, and for irremovable ulcer he has advised gastro-enterostomy combined with jejunostomy. The healing of the ulcer, which is always slow, can be watched by X-ray examination, and he records cures at the end of periods from six months to three and three-quarter years. During this time his patients received neither solid nor fluid food by the mouth.

The question arises as to the need for the gastro-enterostomy. Swallowed saliva and gastric secretion of psychological origin are likely to be the only contents that need to be diverted from the pylorus, unless hypersecretion occurs in such cases apart from digestion. My own impression is that all these activities are so reduced that the stomach is actually put out of action, and the value of the gastro-enterostomy has a bearing on the deformity which may result from cicatrization rather than on the actual healing of the ulcer. I have proceeded on this assumption in a series of 6 cases, of which a few details are given here. In all of them the ulcer was close to the cardiac end of the stomach, adherent, and sufficiently large to be regarded as irremovable. In most of them the stomach was immobile and a gastro-enterostomy on the cardiac side of the ulcer a practical impossibility. There was therefore no choice except partial, and not very partial, gastrectomy, and the much less dangerous operation of jejunostomy.

Two of these cases have died since the operation. One died eighteen months afterwards, having been relieved of pain, but never making much progress in health or appearance. The explanation of this was that his ulcer was carcinomatous, a fact which was proved post mortem. The other fatal case was that of a man whose ulcer I suspected of malignancy, but I have no microscopical proof of this. He suffered from glycosuria, and presented a mass near the cardiac end of the stomach, with a large crater adherent to the pancreas. He tired of jejunal feeding after three and a half months, and, because I thought his ulcer malignant, I allowed fluid by the mouth at this time. An X-ray taken before this permission was given showed some spasm of the stomach, but no hold-up of barium. From this time he continued to improve. Six months after the jejunostomy the tube slipped out, and no attempt was made for twenty-four hours to put it back. Then it was impossible. Considerable progress was made after this, and the patient was delighted, for his weight rose from 8 stone at the time of the operation to 12 stone. But cure was not obtained. Nineteen months after the operation he had a sudden and copious hæmorrhage, and death ensued in two days. Two facts are brought out by this case—namely, the falsity of symptoms in the case of gastric ulcer, and the rapidity of closure in a well-formed jejunostomy.

A third case must be regarded as a failure because the patient, a very sensitive woman, declined to continue the jejunal feeding after the fourth month. Her X-ray showed the ulcer lessened in size, and the fistula healed promptly and needed no operation to close the bowel. In her case pain had never been much in evidence, and for this reason she could not appreciate the value of the treatment.

The other three cases are much more satisfactory.

A. C., male. Ten years' history of indigestion, with severe pain, vomiting, and marked emaciation. Hæmatemesis occurred once in 1921. X-ray examination showed a bilocular stomach with a visible ulcer crater. Jejunostomy was performed in February, 1923. He stuck to the jejunal feeding entirely for ten months, during which time he put on 2 stone in weight. Then he was tempted by cold weather to take milk pudding. This did not interfere with his progress, but a small crater could still be visualized radiographically in December, 1923. About this date the tube slipped out and mouth feeding was resumed entirely. His own expression was that he felt as fit as he ever had done in all his life. His X-ray taken in March, 1924, showed a spasm of the stomach lasting for an hour, but no crater could be seen. The last examination in June, 1925, showed a bilocular stomach, but no ulcer. He was then doing a full day's work of ten hours, weighed 11 stone 3 lb. as against 7 stone 5 lb., and was eating all kinds of food. He has reported that all was well up to November of last year.

J. A., male. History of seven years' duration. Jejunal feeding for four months from June to October, 1923. Persistent shadow visible in X-ray taken in May, 1923. X-ray report in October: "no evidence of ulcer". Consistent progress. Weight in April, 1924, 12 stone 4 lb. On full diet and at work as a seaman. Last X-ray report in April, 1924: "the appearance is that of a normal stomach". No return of symptoms up to date.

R. C., male. Nine months' history. Very severe pain. X-ray showed large extrusion ulcer on lesser curve. Operation revealed a large saddle ulcer with a crater $2\frac{1}{2}$ in. across, adherent both to liver and pancreas. Jejunostomy in February, 1924. Immediate cessation of pain and rapid increase in weight; gained 2 stone in three months during strict jejunal feeding. Confessed to taking a little fluid by the mouth four months after the operation. Only a very little allowed. Steady progress. Returned to work as a railway guard five months after the operation. This he is doing now. He is loath to leave out his jejunostomy plug, and I have not sanctioned this, because the X-ray taken in March, 1925, showed evidence of gas still in the ulcer crater, but no barium was held up in it.

In spite of the fact that these patients all defaulted, and yielded to the temptation of mouth drinking before there was X-ray evidence that the ulcer had healed, the results are encouraging. They were all desperate cases, and my experience confirms Moynihan's suggestion that ulcers not amenable to gastrectomy may nevertheless be treated by jejunostomy. Possibly medical treatment without artificial feeding could show similar results, but it seems a pity that physicians are apt to rely on clinical signs, and do not appear to submit their patients to routine X-ray examination, which alone affords conclusive evidence of cure so long as the abdominal wall stands in the way of inspection of the stomach itself. Even epigastric tenderness is unreliable at times, and it may disappear when the X-ray still shows a hold-up of the opaque meal.

Jejunostomy has a further use in connection with gastric ulcers—namely, as a preliminary to gastrectomy. A poorly nourished patient, worn out with

gastric pain, may be made into a good surgical risk by simple jejunal feeding, and the large operation, which in the first instance could not be tolerated, may be undertaken with every prospect of success.

Walker[32] has advocated jejunostomy in the *pernicious vomiting of pregnancy*, and he quotes a successful case, but I think this may be regarded as rather controversial ground.

Jejunostomy in Intestinal Obstruction.—In opening the discussion on acute intestinal obstruction at the meeting of the British Medical Association last year, Sir William Taylor considered the treatment according to the stage of illness in which the patient was first seen. The early stage of obstruction need not concern us unless it involves the jejunum directly. Full exploration is permissible, and it is only necessary to deal with the obstructing lesion laid bare to the surgeon's view. The second stage is characterized by distention of bowel. Here the obstruction should be relieved, but in addition Sir William advises a temporary jejunostomy; it may be needed only for twenty-four or forty-eight hours. In the third stage the condition of the patient will not warrant an abdominal exploration. Relief of distention, and that under local anæsthesia, is all that he would permit—later, the obstructing cause may be dealt with.

With this teaching I am fully in agreement, based as it is on the arguments I have already brought forward concerning proteose poisoning from the contents of the upper reaches of the intestinal canal. Operating on patients in the second stage, I have often been disappointed in my effort to empty the small intestine, and at the same time unpleasantly conscious of the risk of infection of the peritoneum. If this process can be satisfactorily set going by a jejunostomy, it is much quicker and safer. Intestinal lavage can also be instituted, for, in between the periods of drainage, glucose and bicarbonate of soda can be let into the bowel. Toxins may be evacuated and toxins may be diluted. Both processes are of advantage to the patient. With regard to the suggested treatment of the third stage, the grave condition demands that something be done, and jejunostomy can be performed quite well under local anæsthesia; thus one of the greatest risks of operating on such a patient can be avoided.

The question which naturally arises is: Why make an opening in the jejunum? Victor Bonney[33] is credited with being the first to insist on the importance of draining at this high level, and his argument is that it is the jejunum which is waterlogged with toxic fluid, whereas the ileum contains much gas as well as fluid. I am not sure that this observation is universally true, but nevertheless, in view of the higher toxicity of the bowel contents at this level, I believe the treatment is sound. In my own cases I have often found that the jejunum at operation may yield little but gas; in a few hours, however, fluid begins to pour forth in quantity.

The value of intestinal drainage in true paralytic ileus was well shown in a paper which I published in the *Annals of Surgery* in January, 1910. This concerned two cases following fracture of the ribs, and the cause of the paralysis, no doubt, was the irritation of the splanchnic nerves. The first case ended fatally in spite of abdominal exploration, which revealed no obstructing lesion. The second was much relieved by intestinal puncture, and

finally cured by tying a winged catheter into the distended small intestine. This was reached through a small mid-line incision just above the umbilicus, performed under local anæsthesia. It was impossible to identify the portion of the small intestine utilized, and at the time I assumed that it was the ileum. I have no doubt now that this operation was in reality a jejunostomy, and its good effect probably lay in the fact that the opening was high up. Siphonage was used to drain the bowel, and peristalsis was re-established in twenty-four hours. In forty-eight hours the contour of the abdomen was normal and the patient quite comfortable. The fæcal fistula continued to discharge for three weeks, but it ultimately healed spontaneously. In this paper I urged the value of ileostomy whatever the primary cause of paralysis, whether peritonitis was present or not. Now I should rather say jejunostomy.

Jejunostomy in Peritonitis.—If we transfer our attention to peritonitis, we shall realize that there may be as much risk to the patient from intestinal toxic absorption as from peritoneal toxæmia, and in many cases it is difficult to be sure by which route the poisons reach the blood-stream. Recent successes claimed for lymphaticostomy would rather point to intestinal absorption as the fatal factor. It is certainly true that fatal cases of peritonitis manifest both true obstruction and paralytic ileus. The former can be caused by bands of very recent formation, not necessarily fibrous, but the mere glueing together and angulation of coils of small intestine. Paralytic ileus means a spread of infection from the serous to the muscular coat of the bowel, or a splanchnic intoxication.

This subject has received considerable attention from Sampson Handley,[34] and 'ileus duplex' formed the subject of his Hunterian Lecture in March, 1915. This name he applies to a paralysis of the bowel below the level of the pelvic brim, that is, the lower coils of the ileum and the terminal portion of the colon. The method of treatment which Handley recommends is a short-circuiting of the ileum, with drainage of the large bowel by means of a cæcostomy. In effect this amounts to internal drainage of the small intestine as contrasted with external drainage. Handley advises an ileocolostomy as a preventive measure, and jejunostomy may be similarly employed. The latter is obviously quicker except that it requires a fresh incision, and time and experience alone can show which gives the better results. My own view is that jejunostomy is worthy of a trial as a curative measure if done early. The short-circuiting operation with drainage of the cæcum seems to me rather a severe tax on a patient whose condition is desperate. It would seem likely that the wise plan would be to employ Handley's treatment where prevention is demanded, and external drainage in those cases where the condition is clearly established. My personal experience is limited to the latter operation, and I must admit disappointments. However, one is often called in too late, and I do not regret not having done the anastomosing operation. Fortunately the number of cases where either is required seems to be on the decrease, and for this I think we have chiefly to thank the practice of early operation for appendicitis.

Closure of a Jejunostomy.—If the jejunostomy is done by the Witzel technique trouble rarely arises. If it is carried out in a more primitive fashion —and time and circumstances may sometimes demand this—it is well to

bring the tube out through a portion of the omentum, so that this may plug the opening when the tube is withdrawn. In this way a fistula may be avoided and the patient saved a further operation for its closure when the danger stage has passed.

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PRE-AURICULAR FISTULÆ.

By F. A. R. STAMMERS, Birmingham.

The object of this paper is to record six cases of pre-auricular fistula which illustrate well the familial and hereditary features of this condition, and exhibit one of the complications they may undergo. The literature is briefly reviewed, and the theories of their development are discussed. The two cases suffering complication were admitted to the General Hospital, Birmingham, under Mr. H. H. Sampson, and I am indebted to him for permission to publish them now.

Case 1.—W. B., age 13, a schoolboy in good general health, came up complaining of an ulcer, the size of a sixpence, situated just in front of the left ear. In the crus helicis of each ear was a tiny hole which was the opening of a short fistula, running downwards and forwards for about $\frac{1}{3}$ in. A fine bristle could be passed down each, the one on the left emerging through the floor of the ulcer. The fistulæ were subcutaneous.

History.—Ever since he could remember he had had a tiny hole in the root of each helix. Just in front of the one on the left side he had also noticed a lump about as large as a split pea. Eight years ago this lump became larger and a discharge was noticed through the tiny hole. The skin over the lump ulcerated, and a scab formed, from beneath which a sticky yellowish discharge has exuded intermittently ever since. He has been treated by various doctors with lotions and fomentations, but always without success.

Mr. H. H. Sampson dealt with the case by excising the fistula and its suppurating area, and the procedure has been completely satisfactory. The boy says that the fistula on the right side has never discharged, but a bead of seropurulent fluid can always be squeezed out.

The serial sections kindly carried out by Dr. F. M. W. Lamb show: "Septic granulation tissue which is in direct communication with a sinus lined by squamous epithelium." Apart from a small pigmented mole on the left side of the face, there is no other abnormality present in this patient.

Case 2.—C. A., age 13, a school-girl in good general health, came to hospital complaining of a discharging ulcer in front of the left ear. Her history was that she had always had a tiny hole in the crus helicis of either side, and from time to time she had noticed a little serous discharge from either fistula. A year ago the one on the left side seemed to become blocked up, and a small lump appeared just in front and below the hole. This became larger, and the overlying skin soon broke down and a scab appeared. Sero-pus constantly exuded from beneath this, and fomentations and lotions had failed to remedy the condition. On examination, a fine bristle could be passed through the holes downwards and forwards subcutaneously for about $\frac{1}{3}$ in., the one on the left side emerging through the ulcerating area.

Mr. H. H. Sampson excised the fistula and ulcer with complete success.

Dr. F. M. W. Lamb reports as follows: "The tissue exhibits a cystic space lined by squamous epithelium and containing a substance like mucus. In one part of the section is a nodule of cartilage." This nodule may be a piece of the spina helicis, or a piece of isolated cartilage in an abnormal position, giving support to the embryological explanation favoured in this article.

Each of these two cases has an interesting family history. In *Case* 1 the mother, the maternal grandfather, and the son of the mother's sister have all uncomplicated pre-auricular fistulæ, either bilateral or unilateral. The direction of the track in each case is downwards and forwards, and the opening of the fistula is in the crus helicis. It is said that at the age of 18 the grandfather had an ulcer in front of one ear, but that this cleared up without surgical treatment. The mother has it on the right side only and no discharge can be squeezed out, though a bristle can be passed down the track. The cousin has it on the left side only; a bristle can be passed down a distance of $\frac{1}{3}$ in., and a bead of sero-pus can be squeezed out.

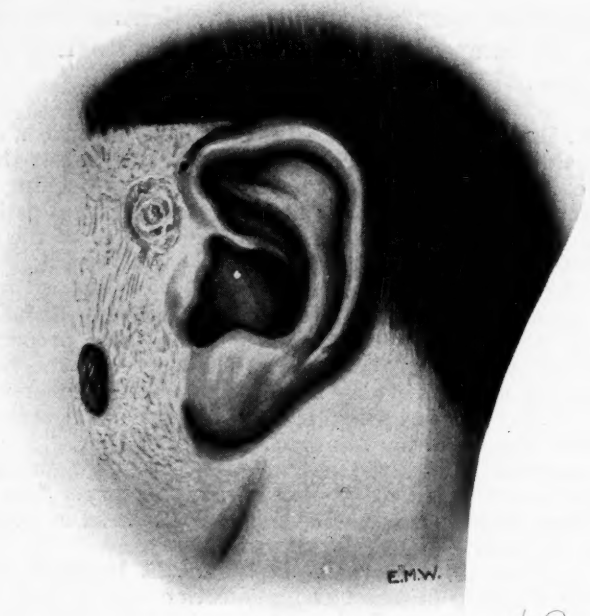

FIG. 207.—*Case* 1. Pre-auricular fistula in left crus helicis, with ulcer below. At the level of the lobule is a pigmented mole.

In *Case* 2 a cousin only is affected, but in her case a mere dimple is present; this is in the crus helicis. No bristle can be passed, and no sero-pus appears on pressing the area in front.

The illustration (*Fig.* 207) shows the condition before operation in *Case* 1. The position of the fistula in the various relatives was identical.

Literature. — Very little notice seems to have been taken of this congenital deformity, and this is hardly to be wondered at in view of the negligible disfigurement and absence of symptoms where no complication ensues. However, when an ulcer develops in front of the ear due to blocking of this little sinus, the presence of which remains unrecognized, and palliative treatment meets with no success, it would seem worth calling attention to this congenital deformity as a possible cause of

Text-books refer to the condition in the scantiest of terms. In 1910 Howell Evans[1] wrote a paper on "Auricular and Peri-auricular Dermoids, Fistulæ, and Tumours of Congenital Origin". He states that these fistulæ are most commonly found "in the region of junction of tragus with crus helicis, of crus helicis with helix, and between antihelix and helix". The six cases reported in this paper belong to the second of these three sites. He also says that these fistulæ tend to run downwards and forwards.

It was Sir James Paget[2] who, in this country, first called attention to these fistulæ in 1878.

In 1909 Sir Arthur Keith[3] gave three demonstrations at the Royal College of Surgeons on congenital malformations of the palate, face, and neck, and stated that there was a close relationship between accessory auricular appendages and pre-auricular fistulæ. He also noted the association of these deformities with other anomalies, such as cleft palate, spina bifida, or imperforate anus. In none of our cases were there any other accompanying aberrations.

Fournier,[4] working in Moure's Clinic, published in 1919 a more complicated type of pre-auricular fistula. This, as in ours, commenced in the crus helicis, but passed downwards through the parotid gland, and opened again on to the surface of the neck just below the angle of the jaw. Moure treated this successfully by excision.

Pritchard, Paget, and Rugani all published cases exhibiting the familial feature of these fistulæ—several cases occurring in each of several families.

With regard to the rarity of the condition, figures seem to vary. Eyle[5] gave 0·26 per cent, and Bezold and Urbantschitsch[6] 0·19 per cent, of the cases coming up to them for examination. Rugani[7] would put it at a much higher figure. I have looked at the ears of some 500 in-patients, and have not seen any case of pre-auricular fistula.

Origin.—Most observers appeal to some developmental error as an explanation of these fistulæ. It may be said that three theories exist: (1) That such a fistula is the remains of the first branchial cleft. (2) That it is due to failure of union between some two of the six tubercles which appear around the posterior end of the first cleft and which go to form the pinna; or some anomalous process occurring in connection with these tubercles. (3) Failure of union of the soft parts corresponding to and overlying the posterior ends of the mandibular and maxillary processes of the first arch. The majority of text-books still favour the first of these three theories, as did authorities like Schwartz, Virchow, and Urbantschitsch. Others, such as His, Gradenigo, and Rugani, state that there is no connection whatsoever between these fistulæ and the first cleft. I have discussed the problem with Dr. Brash, Professor of Anatomy of the University of Birmingham, and he too thinks there can be no connection between the two, and favours the second theory. I would here express my gratitude to Professor Brash for his views on the subject, and references to the literature he has given me.

To make the matter clearer I will briefly describe the development of the external ear and include two of His's diagrams (*Figs.* 208, 209).

The external auditory meatus and concha are developed from the first branchial cleft. Around the posterior end of this cleft six tubercles appear.

Two are on the first arch, three on the second arch, and the remaining one appears at the junction of the first and second, namely, at the posterior end of the cleft. Each one of these six tubercles is destined to form a definite part of the pinna. For convenience of description they are numbered 1 to 6 from before backwards, as shown in the diagrams—No. 3 being the one situated at the posterior end of the first cleft. It will therefore be seen that the posterior end of the first cleft, i.e., that part which goes to form the external auditory meatus, lies between 1 and 2 in front, and 4, 5, and 6 behind, and therefore any vestigial remains of this cleft would lie in a corresponding position. Now, 1 and 2 form the tragus and crus helicis respectively, and 4, 5, and 6 form the antihelix, antitragus, and lobule respectively (His). No. 3 forms the helix. So it will be seen that any deformity resulting from remains of the first cleft will lie between the tragus in front and lobule behind, and will, moreover, communicate with the concha and external auditory meatus. Pre-auricular fistulæ do not lie in this position, nor do they communicate with the external auditory meatus.

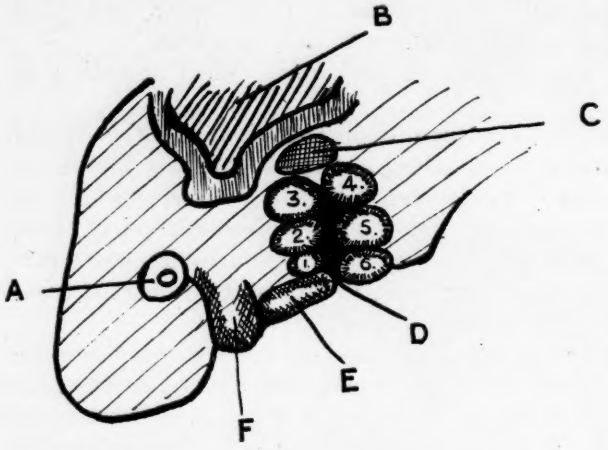

FIG. 208.—Diagram showing: A, Eye; B, Brain; C, Otic vesicle; D, First cleft; E, Mandibular arch; F, Maxillary process.

On the contrary, it is easy to imagine a little subcutaneous track being formed during the coalescence of these six tubercles. The fistulæ of this series of cases were developed in the area of coalescence of 1 and 2. Observations show that the commonest sites are in the crus helicis and between this and the tragus. The downward and forward direction of the fistulæ is explained by the downward and forward development of the mandibular arch with its maxillary process.

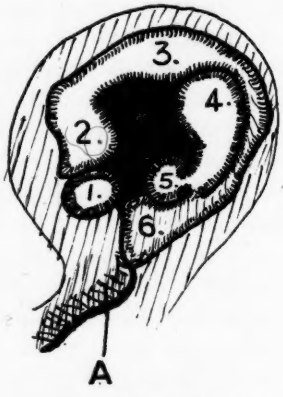

FIG. 209. — Diagram showing; A, Mandible; 1, Tragus; 2. Crus helicis; 3, Helix; 4, Antihelix; 5, Antitragus; 6, Lobule.

Yet a third theory is advanced by some authorities; this is that such fistulæ are the posterior end of a macrostoma. They hold that though the anterior end is normal, so that no actual macrostoma is present, the soft parts overlying the posterior end of the mandibular bar and its maxillary process have just failed to unite. This theory explains the direction of these fistulæ; but a macrostoma, even in its severest form, never extends quite so far back as the ear.

SUMMARY.

1. Six cases of pre-auricular fistulæ are reported. They are all situated in the root of the helix. The six cases came from two different families.

2. Two of these fistulæ had undergone occlusion of the opening with subsequent suppuration.

3. These fistulæ are not remnants of the first branchial cleft, but result from aberrant coalescence of the six tubercles which are destined to form the pinna.

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SHORT NOTES OF RARE OR OBSCURE CASES.

FRACTURE OF THE FIBULA WITHOUT SYMPTOMS.

By SYDNEY SMITH, Cairo.

A young man of 25 years was sent for examination in order to ascertain whether certain marks on his leg had been produced by a firearm projectile, or whether he was attempting to fabricate a case. The injury occurred twenty-three days before examination.

He had a wound on the inner side of the left leg about 1·5 cm. in diameter, and a similar wound on the outer side of the same leg at a lower level. From an examination of the wounds and clothing there was no doubt that he had been shot, and an X-ray photograph was taken to ascertain whether there was another slug in the tissues, for the wounds suggested a fairly rough slug, or slug fired from a shot-gun, rather than a revolver bullet. The skiagram (*Fig.* 210) showed a comminuted fracture of the fibula of considerable extent.

Fig. 210.—Fracture of the fibula without symptoms.

This man had been walking about for twenty-three days, had climbed up two flights of stairs to my office, sat down and rose up repeatedly during the reconstruction of the crime, and yet made no complaint of pain and showed no sign of disability whatsoever.

Although it is quite well known that the fibula plays a very minor part in the support of the leg, I do not think that anyone would have suspected that such an extensive comminuted fracture could have taken place without symptoms. Another point of interest is the very small amount of callus produced during the period.

A CASE OF CERVICAL DISLOCATION AND PARAPLEGIA WITH RECOVERY.

By G. L. ALEXANDER and HERBERT J. SEDDON, London.

In November, 1924, W. C., age 16, was treated at St. Bartholomew's Hospital for multiple infective arthritis. On one occasion he complained of slight stiffness in the neck. He remained in hospital for six months, and continued to attend as an out-patient.

In March and April, 1926, he again complained of stiffness in the neck, but no abnormality was noted until May 28, when a protuberance in the mid-line of the neck behind, with a depression above, was observed. A skiagram (*Fig.* 211) showed that there had been a forward dislocation of the atlas on the axis, with probably a fracture of the odontoid process, but the latter was not visible. No symptoms or signs of compression of the spinal cord were present. Careful inquiry failed to elicit any history of injury other than: (1) A severe blow in the mouth with a cricket bat at the age of 10: the boy states that his neck has been stiff ever since. (2) On coming round from the anæsthetic after the removal of several teeth, he found his head hanging over the end of the table (Jan. 27, 1925).

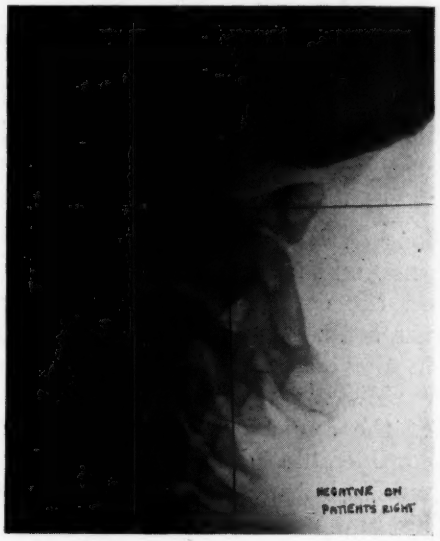

Fig. 211.—Skiagram showing dislocation of the atlas on the axis.

In view of these facts it was considered that dislocation had been present for some time. Accordingly, a plaster cast was taken for the preparation of a suitable support. In the meantime the patient was sent home (May 28, 1926). On June 1 he noticed some weakness of the left arm. On June 7 the left arm was completely paralysed, and the right weak. On the 8th the lower limbs were paralysed. There was no dyspnœa. On the 10th he was brought to hospital and admitted.

On Admission.—Patient was a pale, thin, undersized boy.

Nervous System.—Ptosis of the left upper eyelid; left pupil smaller than right. Other cranial nerves normal. There was flaccid paralysis of both arms except for slight power of pronation and supination on the right side. Intercostals and diaphragm were acting feebly. Both lower limbs were spastic. All active movements were present in the right, and absent in the left except for slight flexion at the hip.

Reflexes: Arms and abdomen absent; knee-jerks exaggerated; right patellar clonus; no ankle-clonus; plantar responses extensor. Sensations

of light touch, heat and cold were lost below the area of supply of C2 on both sides. Painful stimuli were appreciated but not localized.

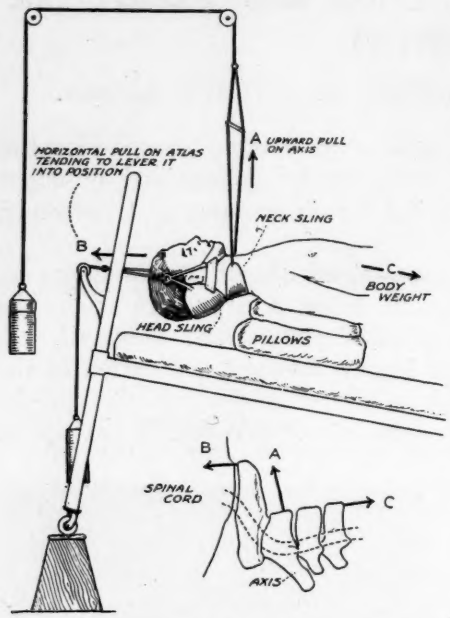

FIG. 212.—Diagram showing the apparatus used in treatment.

There was retention of urine; catheterization appeared to evoke a 'mass reflex' of the lower limbs. There was no dysphagia, alteration in voice, or dyspnœa. The spinous process of the axis was very prominent, and a marked depression existed above.

The boy was put to bed with his head between sandbags. Twenty-four hours later he complained of severe pain in the neck and difficulty in breathing. No action of the diaphragm or the intercostals could be detected, respiration seeming to depend entirely upon the accessory and extraordinary muscles; each respiratory movement was accompanied by flexion of both the hips.

There were three possible lines of treatment: (1) Reduction of the dislocation under an anæsthetic — not advisable on account of the supposed long duration of the lesion. (2) Laminectomy—useless, as the body of the axis, and not its laminæ, was pressing on the cord. (3) Weight extension to the head. This was adopted, as shown in the diagram (*Fig.* 212), which will explain more than a wordy description of the apparatus. The patient found considerable difficulty in swallowing uphill, but gradually became used to his new position.

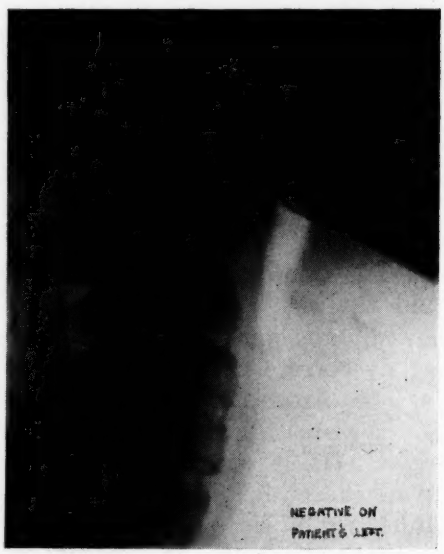

FIG. 213.—Skiagram showing the result of treatment.

After twelve hours the diaphragm was working feebly, intercostals were working well. No dyspnœa. All movements had returned in right lower and left upper limbs. Plantar reflexes extensor. Urine was passed naturally. After thirty-six hours there was well-marked return of power in the left upper limb; but a persistent wrist-drop made it necessary to retain the arm on a cock-up splint.

June 13: Knee- and ankle-jerks were equal and less brisk. No clonus. Plantar responses flexor. Abdominal reflexes present except in left hypogastrium. Sensations normal.

June 14: Counter-extension applied.

June 19: Cock-up splint removed from left arm. No wrist-drop. Swelling in neck smaller.

June 25: X-ray in ward showed dislocation reduced (*Fig.* 213).

At the time of writing the only physical signs are: ptosis of left upper eyelid and constriction of pupil; reflexes brisker on the left side than on the right.

This case owes its interest to: —

1. Its rarity as an isolated lesion.

2. Its obscure etiology. There being no definite history of injury suggests that the dislocation must be pathological, due to cervical caries. Points in support of this are: (*a*) The remote history of injury determining possibly the onset of the disease; (*b*) The chronic recurring attacks of cervical pain; (*c*) The relatively gradual onset of the dislocation and its gradual reduction; (*d*) The apparent destruction of the odontoid process.

Against the diagnosis is the lack of X-ray evidence of caries other than the absence of the odontoid. A pathological process sufficient to destroy the odontoid would almost certainly involve other parts of the vertebræ.

3. The completeness and rapidity of the recovery from such an extensive paralysis.

Our thanks are due to Dr. H. Morley Fletcher for permission to publish the notes of this case.

A CASE OF SPLENIC ANÆMIA, ILLUSTRATING A RARE AND FATAL COMPLICATION OF SPLENECTOMY.

By G. F. WALKER, Leeds.

S. G. R., age 23, was admitted to the General Infirmary at Leeds, and was jointly under the care of Dr. Maxwell Telling and Mr. J. F. Dobson, to whom I am indebted for permission to record the case. The object of this record is to describe an unusual mode of death in splenic anæmia.

The patient stated that he had had no previous illnesses of importance, but complained of three days' abdominal pain, mostly colic, in the left lower quadrant, together with abdominal aching and distention, vomiting, and one brisk hæmatemesis. He was found to have a little free fluid in the abdomen. This increased rather rapidly and then subsided slightly, revealing a peculiar tumour in the left upper quadrant. The tumour was identified as splenic, the organ having an unusually big notch. A blood-count showed: red cells, 3,110,000; hæmoglobin, 30 per cent; colour index, 0·49; leucocytes, 3700.

A diagnosis of splenic anæmia was made, and after preparation the abdomen was opened. At laparotomy several exceedingly large veins were met with in the neighbourhood of the umbilicus. This was the first indication

of the unusually great strain on the collateral circulation between the portal and systemic systems. A further indication of this strain was the discovery that the spleen could not be delivered into the wound because of an extensive plexus of very large veins passing from the posterior abdominal wall to the spleen.

At this point the question of closing the abdomen was considered. It was decided to proceed with the operation. The ubiquitous veins in the ligaments and capsule of the spleen were divided one by one. The pedicle of the spleen, the vessels of which were greatly distended, was secured. The spleen was removed, and the patient did not lose much blood except that contained in the greatly enlarged organ. A few hours after the closure of the abdomen the patient's condition became very bad. He rallied a little, but suddenly had a huge hæmatemesis and fell back dead.

FIG. 214.—Oesophagus with distended veins.

At the post-mortem examination the operation site was found to be perfectly dry and secure. The cause of the hæmatemesis was rupture of an œsophageal vein. The stomach was filled with blood-clot. There was an extravasation of blood into the mediastinum and left pleural cavity from the same source. The liver showed moderate cirrhotic changes.

The accompanying picture (*Fig.* 214) was made shortly after death. It shows the greatly distended veins in the œsophagus. By the closure during splenectomy of many communications between the portal and systemic channels the pressure had been raised in these veins even beyond their capacity.

Although in every similar case of splenectomy the pressure in the collateral circulation must be seriously modified and subject in places to sudden increases, yet I am unable to find on record a case where such sudden increase led to a demonstrable rupture in the œsophageal veins with immediately fatal consequences.

ANEURYSM OF THE ABDOMINAL AORTA: DIAGNOSIS BY A LATERAL RADIOGRAPH OF THE SPINE.

By JAMES F. BRAILSFORD, Birmingham.

The following case is of interest chiefly on account of the fact that, in spite of severe pain and other characteristic symptoms which had been present for two years, whilst radiographs were taken on no fewer than four occasions, the nature of the disease remained undiagnosed until a lateral view of the spine was taken.

History.—T. P., age 47 years. In 1899 he had a venereal sore on the penis, but this was only treated with ointment. He was in the Army from 1898 to 1906, and then rejoined for the whole duration of the War, and was wounded three times. He married in 1910, and had six healthy children. His wife had no miscarriages. In September, 1923, he had pain in the middle of the back at the level of the belt. It was of a gnawing character, with occasional sharp stabs. In April, 1924, he was X-rayed with negative results, and a diagnosis of lumbago was made. In September, 1924, he was again X-rayed, and a slight osteo-arthritis of the spine was deduced from the antero-posterior view.

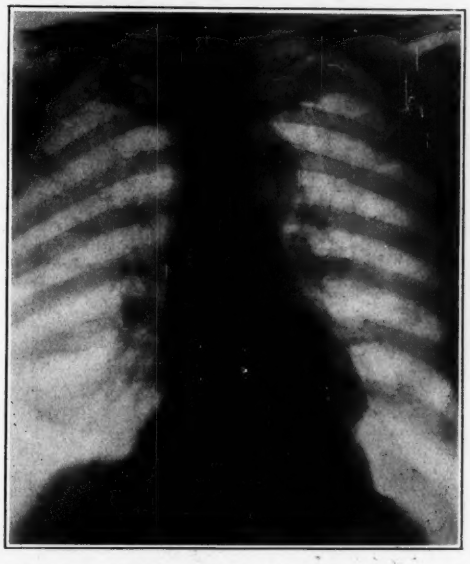

Fig. 215.—Radiograph showing the elevation of the heart shadow.

In January, 1925, he began to suffer from palpitation, and on one occasion about this time he had such severe abdominal pains that he fainted. X rays again showed nothing fresh except some enlargement of the heart. The radiological examination was repeated shortly afterwards, but the report was given that the spine showed nothing except some arthritis. He was advised to work as much as possible, and not to lie up, lest he should become a chronic invalid. In the summer of 1925 he noticed a lump on the left side of the abdomen which was at first regarded as a displaced left kidney. For this condition he was referred to Mr. Billington, who sent him to me for X-ray examination.

On Examination.—Oct. 15, 1925: Patient was intelligent and fairly well nourished. Complained of pain in his back, worse on the right side than on the left, and sometimes shooting down to the front of the thigh. He also had dyspnœa and palpitation on exertion. The abdomen showed a slightly movable tumour about the size of the left kidney below the costal margin. Posteriorly, on the same level as this swelling, some pulsation could be felt,

but there were no sounds on auscultation. The veins in the left lower abdomen and upper thigh were large and prominent. Pulse varied from 100 to 124; regular and of good volume; vessels not hard or thick. Apex beat of the heart ½ in. left of the mid-clavicular line, impulse forcible, no thrill or bruit. Wassermann reaction positive. Marked hyperæsthesia on left side over the area supplied by the 9th dorsal nerve. Impairment of sensation on the left over the area supplied by the 12th dorsal and 1st and 2nd lumbar. Left abdominal reflexes absent.

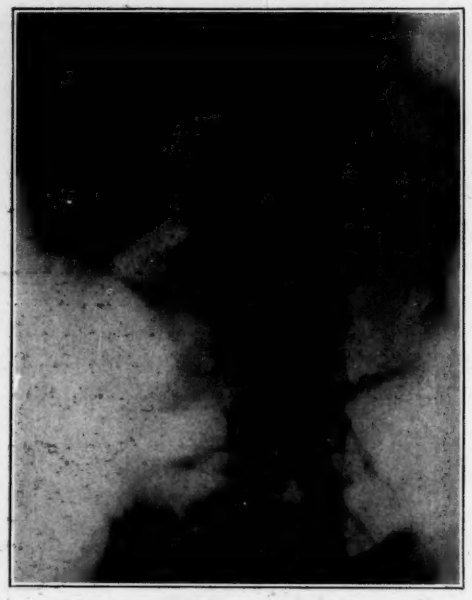

FIG. 216.—Antero-posterior view of spine, showing slight scoliosis.

A radiograph (*Fig.* 215) shows more clearly than the screen the elevation of the heart shadow, which appears to be resting on a body of a little larger size and situated centrally. The thoracic aorta appears to be dilated: this I believe to be a projection effect, as it appeared normal on the screen when the patient was rotated. The lung field appears normal.

Fig. 216 is an antero-posterior radiograph showing a slight scoliosis with its convexity towards the right side at the level of the 12th dorsal and 1st lumbar vertebræ. The left borders of the 12th dorsal and 1st and 2nd lumbar bodies are not so well defined as the borders of the other lumbar vertebræ except at the level of the intervertebral discs. There is some definite bone bridging of the discs between the 11th and 12th dorsal and the 1st lumbar vertebræ on the right side. The left 12th rib is eroded completely through near to the spine, and the head and neck appear to be absorbed.

Fig. 217, a lateral radiograph of the lower dorsal and upper lumbar spine, shows the characteristic 'scolloping' of the 12th dorsal and 1st, 2nd, and 3rd lumbar vertebræ. The intervertebral spacing is not altered. With the production of this radiograph I unhesitatingly diagnosed an aneurysm of the abdominal aorta.

FIG. 217.—Lateral view of lower dorsal and upper lumbar spine, showing 'scolloping' of the vertebræ.

PROGRESS OF THE CASE.—Patient was kept in bed, but continued to

suffer severe pain, with some extension of the area of analgesia. There was no special treatment except by morphia. On several occasions he had severe exacerbations of the pain, which made him faint. On Nov. 29, 1925, he sat up in bed, lit a cigarette, and suddenly fell back dead.

AUTOPSY.—The left side of the chest was full of blood, which had entered it through a rupture of the left dome of the diaphragm. The abdominal aorta presented an aneurysm on its posterior lateral border on the left side at the level of the cœliac axis. The sac had partly ruptured into the sheath of the left psoas muscle. The medial walls of the sac as it lay on each side of the vertebral column were formed by the vertebræ and the intervertebral discs, the bodies of the vertebræ being eroded, leaving the discs almost unaffected. The sac was very thin where it lay against the diaphragm, and at this spot the fatal rupture had occurred.

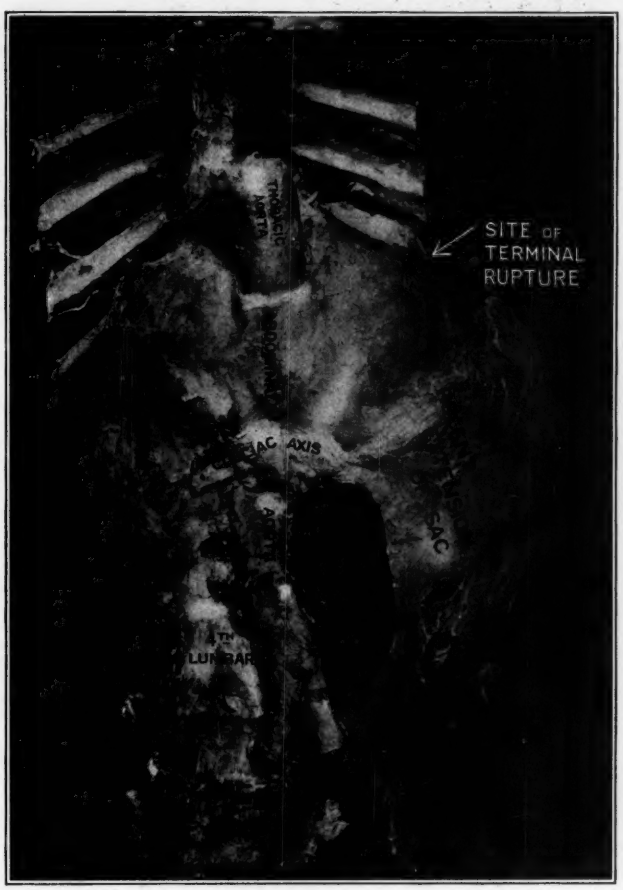

FIG. 218.—Dissection of the aneurysm.

Fig. 218 shows the aneurysm dissected. A window has been cut in the anterior wall on the left side to show the eroded vertebræ. It will be seen that the anterior aspect of the aorta appears to be normal in size.

The patient was shown at a meeting of the Midland Medical Society early in November, 1925, and the specimen illustrated in the photograph at the same Society in December.

I am indebted to Professor W. Billington for his kindness in permitting me to publish the details of this case.

PAPILLOMA OF THE RENAL PELVIS DIAGNOSED BY PYELOGRAPHY.*

By GEOFFREY KEYNES, London.

Villous papilloma of the renal pelvis is to be reckoned among the rarer lesions of the kidney, and very seldom has the diagnosis been arrived at before operation, the kidney having usually been removed on account of a unilateral hæmaturia and the lesion found afterwards. With the more frequent employment of pyelography as a means of investigation, it is probable that the number of instances in which pelvic papilloma has been diagnosed before operation will multiply; one such case has, indeed, been reported recently by Sir John Thomson-Walker.[1] Meanwhile it seems worth while to record the following in detail.

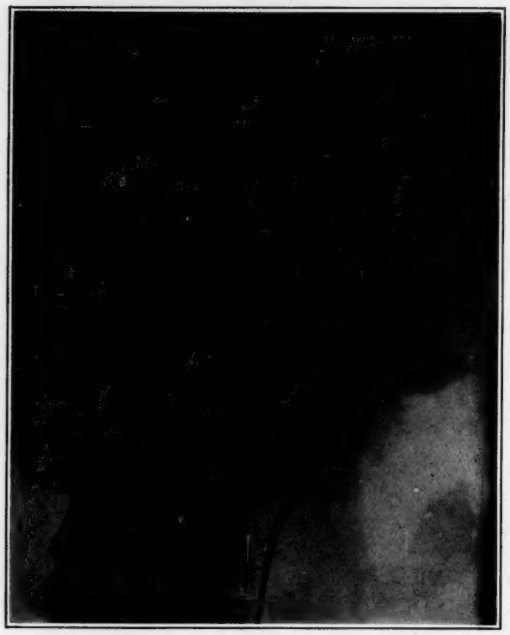

Fig. 219.—Pyelogram, somewhat reduced, showing a filling deficiency in the pelvis, though the calices are filled. The calices are lettered to correspond to the letters on the drawing of the specimen, *Fig.* 220.

The patient, a carpenter, age 44, had an attack of hæmaturia in December, 1924, the bleeding being accompanied by renal and ureteric colic. After the pain had passed off the bleeding ceased, and no further symptoms were complained of for nearly a year. Then, in November, 1925, the patient experienced an aching pain across the back in the lumbar region, and this was accompanied by persistent hæmaturia. There was no increase in frequency of micturition. Five weeks after the return of the bleeding he was admitted to St. Bartholomew's Hospital. The urine then contained a large amount of blood, but no pus, and the man looked anæmic. Cystoscopy showed a normal bladder with a blood-stained efflux of urine from the right ureter. Pyelography was undertaken two days later, and 30 c.c. of 15 per cent sodium bromide solution were injected into the right pelvis without causing the patient any pain. The X-ray photograph showed an appearance which justified a definite diagnosis of papilloma of the pelvis. The inner half of

* From the Surgical Professorial Unit, St. Bartholomew's Hospital, by permission of Professor G. E. Gask.

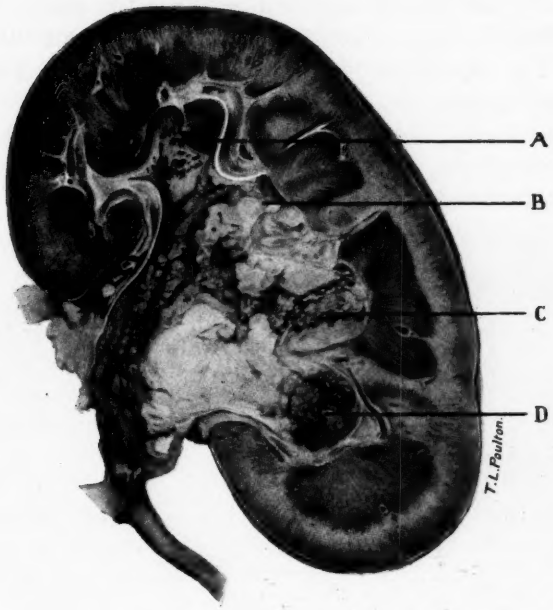

FIG. 220.—Mesial section through the kidney, showing a diffuse papilloma of the pelvis. The main mass of the growth corresponds to the filling deficiency in the pyelogram opposite the lower two calices. The letters correspond to those in *Fig.* 219.

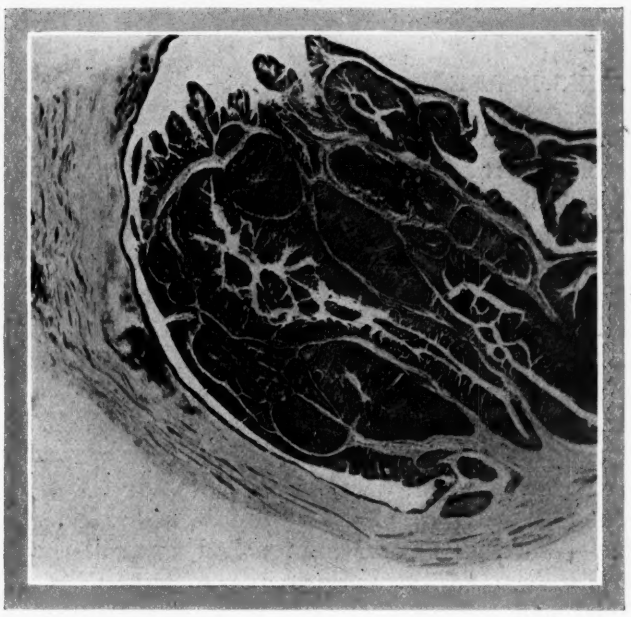

FIG. 221.—Section through part of the growth, including its base. The normal wall of the pelvis is seen on the left. There is no infiltration. (× 15.)

a dilated pelvis was clearly outlined, and all the calices could be seen at the periphery. Between pelvis and calices, however, was a large irregular filling deficiency. The accompanying reproduction of the photograph (*Fig.* 219) is less convincing than the original film, and it has been necessary to outline the denser parts of the shadow in order to demonstrate the deficiency. Clearly no part of the pelvis had been completely obliterated, as by a hypernephroma or carcinoma, since all the calices had been normally filled, and there was no destruction of kidney substance as by an open tuberculous lesion.

The kidney was removed on March 9, 1926, and the condition was found to be as shown in the drawing (*Fig.* 220). The pelvis was partially filled by a diffuse villous growth, the main mass corresponding exactly to the filling deficiency seen in the skiagram. Sections showed the usual structure of a papilloma, and one from the base (*Fig.* 221) demonstrated that it was histologically innocent, no infiltration being found.

One half of the specimen is in the museum of St. Bartholomew's Hospital; the other is at the Royal College of Surgeons.

REFERENCE.

[1] Thomson-Walker, Sir J., *Proc. Roy. Soc. Med.* (Urol. Sect.), 1926, March, xix, No. 5.

OSSIFYING HÆMATOMA OF THE FEMUR, FOLLOWING CONTUSION.

By JAMES RIDDEL and W. CHEYNE WILSON, Plymouth.

Six weeks before admission to hospital, a young man, age 19 years, sustained a severe blow on the front of the right thigh whilst playing football. He continued to play to the end of the game, but during the ensuing few days considerable swelling of the thigh was noticed. As it failed to disperse, and as it interfered with function, advice was sought.

On Examination.—A tumour occupied the antero-lateral aspect of the upper part of the right thigh. Fusiform in outline, about the size of a cocoanut, it lost its definition gradually in the surrounding tissues. Contrary to expectation it was bony hard.

A radiogram (*Fig.* 222), taken by one of us (W. C. W.), disclosed a tumour undergoing ossification which, from the history and the radiological features, was recognized as an ossifying hæmatoma of the upper end of the shaft of the femur.

Operation.—The tumour was approached through the fascia lata, and was found to lie deep to the periosteum of the femur. The walls were half an inch thick and composed of cancellous bony tissue. A central cavity was filled with organizing blood-clot. The tumour was removed except for bony spicules which remained adherent to the periosteum.

Subsequent radiograms showed that ossification was still proceeding in slight degree, as would be expected, but function had been restored and the thigh had resumed its natural form.

Comments.—An ossifying hæmatoma may be mistaken for a sarcoma. The lesion is the result of hæmorrhage beneath the periosteum following traumatism. In children, the condition is commonly associated with scurvy.

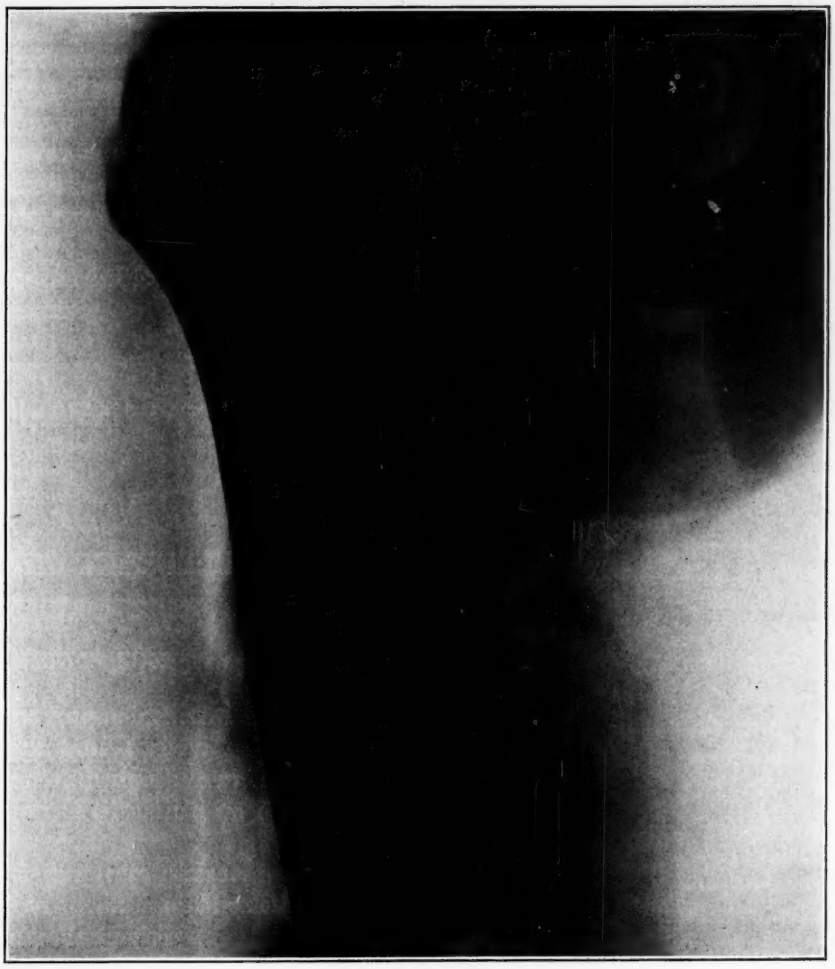

FIG. 222.—Ossifying hæmatoma of femur.

The periosteum in children, being but loosely attached to the shaft, is the more easily stripped from epiphysis to epiphysis. In adults, ossifying hæmatoma is less frequent. The periosteum is more firmly attached, and hæmorrhage is, in consequence, less extensive as a result of traumatism, and tends to be more localized.

Baetjer and Waters[1] describe 17 cases in adults; 16 followed injuries at football and 1 followed the kick of a mule. They all occurred on the

anterior surface of the femur. In nearly half of the cases a clinical diagnosis of sarcoma was made. They stress the following points in differential diagnosis: (1) In hæmatoma there is a definite sharp calcium border, since hæmorrhage is limited by the periosteum. (2) There is no involvement of bone, and the calcium salts are laid down roughly parallel to the shaft. (3) In malignant bone tumours there are no sharp borders, the calcium salts are laid down more or less perpendicular to the shaft, and the bone is frequently involved.

REFERENCE.

[1] BAETJER, FREDERICK H., and WATERS, CHARLES A., *Injuries and Diseases of the Bones and Joints* (1921, Lewis), 277.

REVIEWS AND NOTICES OF BOOKS.

Manual of Surgery. By the late ALEXIS THOMSON, M.D., F.R.C.S. Ed. and Eng., Professor of Surgery, University of Edinburgh; and ALEXANDER MILES, M.D., LL.D., F.R.C.S. Ed., Consulting Surgeon, Edinburgh Royal Infirmary. Seventh edition. Crown 8vo. Vol. I, pp. 565 + xiv, with 169 illustrations; Vol. II, pp. 663 + xvi, with 299 illustrations. 1926. London: Humphrey Milford. Each 12s. 6d. net.

THIS popular Edinburgh text-book is too well known to require any detailed review. The present edition does not present any marked alteration as compared with the last in these two first volumes. The work is specially notable for the number and excellence of the illustrations, and the neat, handy volumes into which the book is divided. Operative surgery is relegated to a separate volume, and the absence of description of operations as part of the treatment of various surgical conditions has both advantages and drawbacks. In such matters, for example, as the treatment of fractures and cleft palate it is difficult to present the problem fairly unless some description and illustration of operations are given. We think that a more complete account might have been given of infections of the fingers and hand on the lines of Kanavel's work. The figure (145) illustrating the X-ray appearance of a myeloma is poor and not at all typical. We think that the statement that the treatment of osteitis fibrosa is merely protective requires amplification.

In describing treatment of cancer of the tongue it is stated that the tongue should be removed by the thermocautery; but no mention is made of diathermy, which surely is an easier and a much more thorough method which causes very little pain.

Speaking generally this manual is an excellent description of surgical diseases, but it is rather deficient as a guide to treatment.

Young's Practice of Urology: Based on a Study of 1,500 Cases. By HUGH H. YOUNG and DAVID M. DAVIS; with the collaboration of FRANKLIN P. JOHNSON. 2 vols. 8vo. Pp. 1433, with 1010 illustrations, 20 in colours, by William P. Didusch. 1926. London and Philadelphia: W. B. Saunders Company, Ltd. £5 5s. net.

THIS monumental work is based on the study of 12,500 histories of urologic cases taken from the files of the James Buchanan Brady Urologic Institute, and completed by a thorough and elaborate 'follow-up' system. Its great value is that it represents the personal effort of a surgeon universally recognized as one of the chief authorities on urology, and everywhere does it bear the stamp of marked originality—though full consideration and appropriate recognition are given to the work of others. Every branch of the subject is treated in a thoroughly practical fashion, and although theoretical questions are fully discussed whenever they have any practical bearing, yet practicability remains throughout as the outstanding feature. The general arrangement of the subject has been carried out mainly on a pathological basis, the authors rightly considering a thorough knowledge of the pathology of each condition dealt with as essential to the proper understanding of its treatment. The detailed description of the technique of surgical procedures is separately considered in the second volume, much unnecessary repetition being thereby avoided.

The opening chapter deals with the physiology and pathology of micturition, and incorporates much original work of Dr. Young and his assistants. The relation

of the 'trigonal' muscle to Mercier's bar and the uvula vesicæ is fully described, and the important part played by this muscle in *actively* opening the internal sphincter during the act of micturition is strongly insisted upon. A very complete account is given of the neurologic lesions of the bladder and of the troubles of micturition consequent upon tabes and other diseases of the spinal cord.

The second chapter, one of the best, deals with the 'obstructive uropathy'. The results of recent research upon the production of hydronephrosis by ligature of the ureter combined with partial obstruction of the renal artery or vein indicate that arterial obstruction hastens the progress of hydronephrotic atrophy: the renal atrophy consequent upon renal back-pressure is only in small part due to direct pressure upon the renal cells, the main cause being pressure-interference with the blood-supply. Diverticula of the bladder, congenital and acquired, are excellently described and illustrated. Laboratory tests for the estimation of renal function receive very special attention, and a new instrument—the phthaleinometer—is presented for the rapid estimation of the drug excreted in the phenolsulphonephthalein test, which of all the excretory tests is that most strongly recommended. The value of laboratory tests of renal function is thoroughly discussed; the judicious conclusions arrived at are: (1) They supplement but do not replace observation of the patient; (2) The additional information obtained from laboratory tests is indispensable; (3) No single laboratory test proves anything; it is merely another element in the complete clinical picture we are trying to construct. In the treatment of obstructive uropathy the guiding principles are clearly and concisely enunciated, and the importance of avoiding operation until the renal function has reached its 'optimum' level is rightly insisted upon. "The future of the patient is decided much more, at the present time, by the care and skill displayed in the ward before operation than it is in the operating room itself". In cases of chronic vesical distention with long-standing renal back-pressure, the great danger of a too sudden relief of this pressure is strongly emphasized, and an account is given of the Young-Shaw apparatus by which a very gradual decompression can be safely achieved.

A special feature of the third chapter—on general urogenital infections—is the full description of the special technique for urologic bacteriology, including the collection of specimens, dried smears, staining methods, and culture media. The pathology, symptoms, diagnosis, and non-operative treatment of all the general infective diseases of the urogenital tract are fully described, particularly excellent being the account of those due to the gonococcus and the *B. typhosus*. Since 1917 extensive researches have been conducted at the Brady Urologic Institute in the field of urinary antiseptics, and some 265 different combinations of germicides with phenolsulphonephthalein have been tested, of which No. 220—'mercurochrome'—and No. 253—'meroxyl'—are advocated. Mercurochrome has been largely used as a local antiseptic to wounds and mucous membranes, intravenously in cases of septicæmia from colon bacillus, staphylococcic, and streptococcic infection, and in the treatment of pneumonia, genito-urinary infections, gonorrhœal urethritis and arthritis, furunculosis, cellulitis, and abscess. It is injected intravenously in 1 per cent solution up to 5 to 10 mgrm. per kilo. of body-weight. The results are given in tabular form, and many striking and beneficial effects are claimed; so striking, in fact, that it is suggested they cannot be due entirely to the direct destructive or inhibitive action of the drug, but are largely the effect of the indirect stimulation of the defences of the body.

In the section on tuberculosis it is held that the vesiculæ seminales are the primary and principal seat of the infection, and that the epididymis is usually involved by lymphatic extension along the vas deferens, the globus minor being first involved. The results of simple castration and epididymectomy are stated to be very poor, and, in consequence, the operative treatment recommended for tuberculous epididymitis is extremely radical—consisting of removal of the seminal vesicles, ampullæ, and both lateral lobes of the prostate through the perineum, after which the epididymis (and testicle also, if involved) and vas deferens are extracted through an incision in the groin (or both groins in bilateral cases).

In the chapter on 'urolithiasis' we would draw attention especially to the

excellent account of Kelly's 'wax-tip' method in the diagnosis of renal and ureteral calculi, and also to the indications for operative treatment in bilateral renal calculi.

The authors do not enter into any discussion as to the essential nature of 'benign hypertrophy of the prostate', but are content with the statement of their belief that the process is a hyperplastic and not a neoplastic one. They accept the view of Motz and Perearneau that all these hypertrophies arise from the mucosal or suburethral glands. Their records show that, of prostates which cause obstruction, 20 per cent are carcinomatous, and of these fully half are hypertrophies associated with carcinoma. The special cystoscopic technique necessary in benign hypertrophy is very fully detailed, as is also its pre-operative treatment. Dr. Young shows that radical suprapubic prostatectomy had been carried out in America many years before Freyer operated upon his first case. The operative treatment advised for benign hypertrophy is Young's conservative perineal prostatectomy; this has been performed in the Clinic 1049 times, with 36 deaths, a mortality of 3·4 per cent; whereas for 108 suprapubic prostatectomies the mortality was 8·2 per cent.

In the chapter on neoplasms, which completes the first volume, the term 'nephroma' is preferred to 'hypernephroma', since it does not commit one to the unproved theory of its adrenal origin. In malignant renal tumours the transperitoneal operation is preferred, and a wide removal of the perinephric fat and of the peritoneum covering the anterior surface of the kidney is advised. The use of radium and X rays in the treatment of malignant disease of the kidney, bladder, and prostate is exhaustively detailed, and many special radium applicators are described.

The second volume opens with a very complete account of the malformations and abnormalities of the urogenital tract, including a case of true lateral hermaphroditism. Chapter XII—on the diagnostic significance of special urologic symptoms—will prove extremely useful to the general practitioner, and that following it on the examination of the urologic patient is of great value to both practitioner and specialist. The rest of the volume is devoted to the detailed technique of operations upon the genito-urinary tract, the various procedures being very completely described and illustrated. In addition to the operative procedures already referred to, mention must be made of Young's original operations for the radical treatment of cancer of the prostate, of malignant growth of the testis, of epithelioma of the penis, of vesical diverticula, and of the 'punch' operation for contracture of the vesical orifice. Total cystectomy has never been done at the Brady Clinic, and is not considered justifiable. Many original instruments are referred to, including Young's well-known prostatic tractor, cystoscopic rongeur, evacuating cystoscopic lithotrite, prostatic punch, and automatic perineal elevator.

The work is extremely well and artistically illustrated with over 1000 figures, mostly original, and 20 beautifully coloured plates. It constitutes the most complete, original, and practical description of urologic surgery yet produced, and will undoubtedly take its place as the standard authority thereon.

Carbohydrate Metabolism and Insulin. By JOHN JAMES RICKARD MACLEOD, F.R.S., M.B., LL.D. (Abdn.), D.Sc. (Hon.) (Toronto), Professor of Physiology, University of Toronto. (Monographs on Physiology.) Demy 8vo. Pp. 357 + xii, with 33 illustrations. 1926. London: Longmans, Green & Co. 18s. net.

THIS book, the latest member of the series of *Monographs on Physiology*, is essentially an account of experimental work carried out in attempts to elucidate the nature and mechanism of the changes occurring during the metabolism of carbohydrates.

The discovery of insulin and its well-known effects offered a new means of experimental study of these problems, and it is with work leading up to, and arising out of, this discovery that the author is especially concerned. Hence he begins by tracing the development of the view that the islets of Langerhans are responsible for a specific internal secretion, and so leads up to the preparation of insulin. Then

follows an account of experimental work in which the effect of insulin on normal and diabetic animals is noted with reference to the various criteria which have been used in the study of sugar metabolism. Finally, he discusses the mechanism of the action of insulin in the light of these results.

It will be noted that the book is in no sense a treatise on diabetes. Hence it makes no direct appeal to the clinician. Nevertheless a careful reading of it will not fail to add something of interest and value to his understanding of this disease. Especially may this be said of the chapters on hyperglycæmia and hypoglycæmia. The surgeon will find interest in the account of hyperglycæmia following general anæsthesia and the effect of insulin in preventing and modifying it; but its possible relation to post-operative acidosis is not dealt with.

Undoubtedly the book's chief appeal is to the experimentalist, since it gives with considerable care and patience a summary of the work done in this important field, together with an attempt to assess the value of the results obtained. Moreover, it shows that many problems are still unsolved, and thus indicates the lines along which further useful work may be attempted. Its value in this respect is enhanced by the very useful bibliography following each chapter.

Praktikum der zahnärztlichen Chirurgie. By Dr. Bernhard Steiner, Demonstrator at the Dental Institute of the University of Vienna. Demy 8vo. Pp. 199, with 113 illustrations and 2 plates. 1926. Vienna and Berlin: Urban & Schwarzenberg. M. 9; bound, M. 10.80.

This book, as the title indicates, deals with the surgical aspect of dental surgery, and is particularly welcome for that reason, as there are very few books which embrace the subjects dealt with in this volume. These include extractions, resection of roots, cysts, and replantation, with chapters on anatomy and anæsthesia.

On the subject of anæsthetics the author clearly indicates his choice of local anæsthesia whenever possible, using a freshly-made novocain-adrenalin solution varying from 1 to 4 per cent. He mentions that the firm of Woelm are introducing ampoules in which the powder and liquid are separated by an easily-fused metal plug, which when fused causes the powder and liquid to mix and form a fresh solution. This certainly appears to be a convenient and time-saving device for use in the surgery. In describing the details of the use of local anæsthetics, the author points out his preference for submucous injections when dealing with the maxilla, and gives a word of warning against inserting the needle under the periosteum. He further states that block anæsthesia for the maxilla is not to be encouraged, and should be used only in cases of absolute necessity. For mandibular anæsthesia he favours inferior dental injections, and describes a method of external injection for use in cases of infection or inability to open the mouth. Regarding general anæsthesia, he lays particular stress upon the importance of ethyl chloride, given by the the open method, in preference to nitrous oxide, and quotes Levy as having given 17,000 cases without a fatal result.

The chapter on extractions is very interesting, and is well illustrated. The author divides the cases into typical and exceptional, the methods used in the former being similar to those most generally adopted in this country. In dealing with the exceptional cases, he mentions many ingenious methods of removing buried roots, etc., and gives a very clear description of his technique for the removal of impacted lower wisdom teeth, showing a preference, when dealing with severe impactions, for first cutting off the crown and then removing the root separately.

The subject of apicectomy is fully described and illustrated. The author's technique is first to fill the canal, and then to make a sufficient opening to expose the apex, and, if necessary, to refill the cut surface of the root with amalgam. He rightly stresses the importance of leaving a sloping surface of cut bone in order to permit drainage.

In the chapter dealing with cysts, the illustrations show very clearly how cysts in the maxilla invaginate into the antrum without perforating it, and also how they

are able to push their way into the nasal cavities and displace the nasal structures. In treating the operative aspect, the author advocates maintaining the orifice open by a vulcanite plate and permitting the cavity to granulate up from the base.

The remaining chapters on injuries, unerupted teeth, and epulis are disappointingly short, especially that dealing with unerupted teeth.

The book forms a very interesting addition to the library; although one may not agree with some of the operative details, it is very useful in presenting methods adopted in other countries, and well repays a careful study.

La Gastroscopie: Etude clinique et expérimentale. By JEAN RACHET, ancien Interne des Hôpitaux de Paris. Medium 8vo. Pp. 120, with 36 figures in the text and 10 plates, of which 2 are in colours. 1926. Paris: Gaston Doin et Cie. Fr. 20.

THIS is a clinical and experimental inquiry whether or not gastroscopy has a right of place in the methods of investigation of diseases of the stomach, since, although introduced more than fifty years ago, much less progress has been made therein than in all the other 'endoscopies'. In the opening chapter the author reviews the history of his subject and the various types of gastroscope that have from time to time been advocated, and decides in favour of an instrument which is straight rather than curved, rigid rather than flexible, with indirect rather than direct vision, capable of being introduced over a metallic conducting thread previously swallowed as a guide rather than blindly passed, and of comparatively small bore (27, Charrière scale). Such is the gastroscope of his chief, Dr. Benaude, and this the author has adopted in his work: the instrument and the detailed technique of its use are fully described. After stating very fairly the indications and contra-indications of gastroscopy, the conclusion arrived at is that the method, not devoid of danger, is for exceptional use only, and should never be allowed to replace other modes of gastric investigation—chemical, radiological, etc. When these fail to yield any conclusive evidence, gastroscopy should be resorted to as a possible substitute for exploratory laparotomy, but only where the services of a specialist, skilled in this procedure, are available. Moreover, since even under the most favourable conditions it is impossible to explore the whole of the gastric cavity, a negative finding does not necessarily exclude the presence of a pathological condition.

The technique of gastroscopy is described in great detail. General anæsthesia should not be used, and the local application of a 10 per cent solution of cocaine to the pharyngo-laryngeal region is recommended in combination with scopolamine and morphine hypodermically. The position of the patient during gastroscopy is very thoroughly considered from the two points of view of (1) ease of introduction of the instrument, and (2) the visibility of the gastric cavity, and it is in this connection that the results of the author's important anatomical and radiological researches in the living and on the cadaver are given. He concludes in favour of the left lateral decubitus as the routine position for clinical use: in the cadaver he has found that 'dorso-lumbar lordosis' simplifies intragastric vision, but he has not, as yet, had sufficient practice with this posture in the living subject to enable him to decide as to its advantages. The normal gastroscopic appearances are well described, and the importance of a recognition of the pylorus and of the lesser curvature is duly insisted upon. In gastric ulcer the method is particularly useful in the diagnosis of recent and superficial ulcers, which do not yield any radiological evidence of their presence: it reveals that around the actual ulcer there is, for a varying but often considerable distance, a zone of 'gastritis', where the mucosa is swollen, congested, very vascular, and frequently the seat of numerous small superficial erosions. The early diagnosis of cancer of the stomach should be assisted by gastroscopy, but, as yet, only cases where the disease is already advanced have actually been examined: the recognition of 'ulcer-cancer' is scarcely probable by gastroscopy when one considers its great difficulty even after operative exposure.

The work is well illustrated throughout, and is a very complete and impartial statement of the present position of gastroscopy as a method of clinical investigation.

The Cancer Review: A Journal of Abstracts, issued under the Direction of the British Empire Cancer Campaign. General Editor: FRANCIS CAVERS, D.Sc., M.R.C.S. Vol. I, No. 1. Royal 8vo. Pp. 80 + viii, illustrated. 1926. Bristol: John Wright & Sons Ltd. 30s. per annum (10 numbers); single numbers, 3s. 6d. net.

BY a sad coincidence the death of Sir William Leishman, to whose initiative this new journal largely owes its existence, has preceded the issue of the first number.

The Cancer Review is issued under the auspices of the British Empire Cancer Campaign, and its object is the publication of abstracts and views of the current contributions to the ever-increasing literature on cancer. The idea is to include in the *Review* an abstract of every important publication bearing on the problems of cancer research in the widest sense. Clinical papers, as well as purely pathological ones, are included in the scope of the *Review*. It is evident, therefore, that it will be indispensable to all surgeons who wish to keep abreast of current knowledge of cancer, as well as to pathologists.

The *Review* is under the General Editorship of Francis Cavers, D.Sc., M.R.C.S., assisted by an able Editorial Committee which is composed of well-known pathologists and clinicians who are especially interested in the study of the disease.

This first number contains 127 abstracts derived from the literature of the world. It is printed in large clear type on good paper which enables a reader in the course of an hour or two's study to find out what is being done at the moment by research workers everywhere. The editorial work has been carefully done, and brevity has been attained without any loss of intelligibility. The publication of such a journal cannot fail to stimulate research, to prevent waste of effort and overlapping, and to assist clinicians in treating the disease.

The British Empire Cancer Campaign, in establishing the journal, has performed a notable public service.

The Surgery of Gastro-duodenal Ulceration. By CHARLES A. PANNETT, B.Sc., M.D. (Lond.), F.R.C.S. (Eng.), Professor of Surgery in the University of London, Surgeon to St. Mary's Hospital. Demy 8vo. Pp. 154, illustrated. 1926. London. Humphrey Milford, Oxford University Press. 10s. 6d. net.

IT would be impossible in a short book, such as this is, to deal with the subject of gastro-duodenal ulceration exhaustively, and the author must be congratulated on the concise manner in which he has placed before the reader what he describes in his preface as a fair view of the problems which confront the surgeon who seeks to bring relief to the victims of this crippling disease.

We seem to have got very little further in the pathology and etiology of ulceration, and in the first two chapters the author contents himself with shortly epitomizing the most authoritative views under these two headings. One or two points may be mentioned as of some importance. The first is the statement that the commonest situation for a duodenal ulcer is the pancreatic wall at the junction of the first and second parts. We do not think this will meet with general acceptance. Again, as to the recognition of a duodenal ulcer on the operating table, the author states that the pyloric vein is not a sufficiently reliable guide, and he finds it difficult to differentiate sometimes between duodenal and gastric ulcers in consequence. We think there can seldom be any difficulty in this matter if the white line of the pylorus be observed as well as the pyloric vein, for these two bear a very constant relationship to each other. A third point is the question of malignant change in gastric ulcers. This is a very difficult subject, for the evidence of malignant disease recognized by some is strenuously opposed by others, especially that which consists of detached epithelial cells; the former regard these as definite evidence of a malignant transformation, whereas the latter are equally convinced that they are cells isolated by fibrous tissue during attempts at healing. As the author says, if these are evidence of a malignant change, it is a curious thing, considering that they are observed in upwards of 70 per cent of cases, that cancer is not seen with great frequency after treating the gastric ulcer by gastro-enterostomy alone. We think he sums up the matter very fairly when he says, "it therefore seems clear that whilst

the likelihood of cancer development is not nearly so great as some American surgeons believe, yet it is a distinct danger to be reckoned with, and it suggests that it is always preferable to resect a simple ulcer of the stomach whenever the operation is not contra-indicated on technical or some other equally adequate grounds."

In Chapter 3 symptomatology is discussed. There is nothing new in this section. The author says that the first symptom of a chronic gastric ulcer may be perforation or hæmorrhage. We think this must be a very rare event; a careful inquiry will almost always elicit the previous existence of symptoms, and we do not agree that duodenal ulcer is difficult to diagnose clinically; on the contrary, it is one of the easiest abdominal lesions to be sure of, if the earliest periods of the patient's complaint of symptoms be thoroughly investigated; later the character of the symptoms changes, and an anamnesis of this period alone would certainly lead to confusion.

Chapters 4, 5, and 6 deal with the surgical treatment of ulcer. In the first of these the physiological effects of the operations usually performed are considered. This is quite a good chapter, but as the physiology of the normal stomach is still a matter of contention, it is not surprising that there is no unanimity on the physiological aspects of the post-operative functions. The old controversy as to the mechanical or so-called physiological action of gastrojejunostomy is dealt with, and though the author seems to belong to the latter school of thought, we do not think he establishes his position any too securely.

With regard to the operations performed for gastric ulcer, we find ourselves in complete agreement with the author when he says that an operation such as gastro-enterostomy, which admittedly gives less satisfactory results than those which deal with the ulcer directly, should not be performed as a routine because in the ordinary surgeon's hands it is safer. If a surgeon cannot acquire the technique to perform resection with a risk only negligibly greater than that of gastrojejunostomy, he should not undertake to operate on unperforated gastric ulcers. The author reviews the various operations performed, giving statistics of cures from authoritative sources. These, like all statistics, need to be accepted with caution. He prefers sleeve resection or Péan's operation (also called Billroth I) according to the circumstances existing, and claims that in his hands the results are better than those following resections of the Polya type.

In Chapter 6 the operative treatment of duodenal ulcer is considered. The alternatives given are gastrojejunostomy and partial duodenectomy, of which the author prefers the latter. It is a little difficult to see why, as his death-rate is 6·8 per cent in 29 cases, a mortality four times that of gastrojejunostomy. It is true the after-results of the latter operation are not ideal, but it remains to be seen whether those of duodenectomy are any better. Up to the present there seems little to choose between the operations in this respect. We regret that the author does not mention the operation of gastroduodenostomy, an operation of which most surgeons speak highly in the few cases in which they perform it, and which is attended by after-results as nearly perfect as can be.

Hæmorrhage and perforation are dealt with in Chapters 7 and 8. There is nothing new in these sections. We note that amongst the symptoms of the latter initial shock is mentioned, as it is in most text-books. This generally accepted notion is, we believe, inaccurate, as a pulse-rate of 80 or thereabouts will be found almost always in the pre-peritonitis stage.

Chapter 9 is devoted to the description of the technique employed in the operations performed. With one or two exceptions this is as the text-books describe it. We note that emphasis is laid on retaining all the mucosa exposed after opening the stomach and jejunum during the operation of gastrojejunostomy. Also, when the ulcer is fixed to the pancreas, no attempt is made to remove the base, and the adherent necrotic tissue is not treated in any way. The author says he has not seen any ill effects result from this policy. On the other hand, we have never seen any harm caused by shaving the pancreas, and a clean removal appeals to us as better surgery.

The final chapter is given to the consideration of the post-operative complications. Those which appear in the text-books are deftly dealt with, but in addition

there is a paragraph on " injuries to the bile and pancreatic ducts in duodenectomy ". The author states there is a great risk of this occurring, and that it has happened in a certain proportion of cases, and adds, of course, that it is a serious matter. The results of an operation which involves this risk must be shown to be much better than those after gastro-enterostomy before it can be recommended as an alternative treatment. This book gives us the impression that the author is striving to establish this position, but we feel that he is not very convincing. There seems little to choose in the late results. Removal of the duodenal ulcer of course eliminates the chance of perforation of, or hæmorrhage from, it in after years, but these are rare events after gastrojejunostomy, and the immediate risks of duodenectomy are undoubtedly greater than those of the former operation. Moreover, it is not yet established that the fear of an anastomotic ulcer disappears ; indeed, for all we know at present there may be a greater incidence. However, the operation is being given a trial, and no doubt for a while will become increasingly popular, and the book is well worth reading from this aspect.

References to unsettled problems are given at the end of each chapter, and instructive illustrations appear throughout the book.

Though there is much in the volume of which we do not approve, we think it is one which every surgeon should read. It not only gives the views and opinions of the author (and it is always refreshing and instructive to have these from the pen of an expert), but it contains those of all the leading surgeons and many experimentalists, expressed in a neat and concise manner, and also much statistical information.